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PULMONARY TUBERCULOSIS



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# Pulmonary Tuberculosis

Its Diagnosis and Treatment

A HANDBOOK FOR STUDENTS AND  
GENERAL PRACTITIONERS

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FIFTY-ONE ILLUSTRATIONS

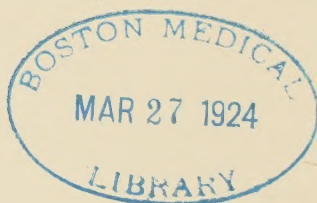
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## PREFACE

THE object which I had in view while writing this manual was to present in short, concise form the salient points in connection with Pulmonary Tuberculosis.

Considerable difficulty was felt in selecting the material for inclusion because of the wealth of knowledge we now have of the disease and the enormous literature which exists on the subject. In order to keep the volume within certain prescribed limits I have had to refrain from discussing many points of much interest, deeming them non-essential for the purpose in view.

The book is written as an introduction primarily for the student, but it is hoped that it may be of some value to the general practitioner who desires to refresh his knowledge in a most interesting and variable disease.

I have occupied some space in dealing with the physical signs of the chest, because this, as I have found by experience in teaching, is the weak point of the student, and because of the importance of the physical signs in the diagnosis of early tuberculosis.

I have to express my indebtedness to Dr Robert Knox for the X-ray photographs, and to Messrs Maclehose for the use of Fig. 43. To Dr MacGregor I owe much for his helpful criticism, and to Dr Linklater for his assistance in reading the proofs. The invariable courtesy of the publishers made my task one of pleasure.

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# PULMONARY TUBERCULOSIS

## CHAPTER I

### INTRODUCTORY

PULMONARY Tuberculosis is one of the oldest of recognised diseases that afflict humanity. We have proof of its presence in pre-historic days. Amid the tombs of Egypt, bones have been discovered showing the results of tuberculous disease among people who died two or three thousand years before Christ. From these and other evidences we conclude that this disease in the remote past was not uncommon. Probably in those days, as in these, tuberculosis was an expression of an imperfect civilisation; yet that seventeenth century poet and physician, Sir Thomas Browne, tells us that "some think there were few consumptives in the old world, when men lived much upon milk, and that the ancient inhabitants of this island were less troubled with coughs when they were naked and slept in caves and woods, than men now in chambers and feather-beds."

Even Sir Thomas Browne in his *Religio Medici* confesses that "in our study of anatomy there is a mass of mysterious philosophy, and such as reduced the very heathens to civinity." One thinks of the gold and ivory statue of Æsculapius with the serpent twined round his staff. No wonder that from these twilight years no ray of medical learning has come down to us, and it is only when we come to the writings of Hippocrates (460-377 B.C.) that we get an idea of the extent of the medical knowledge of the disease. From these writings it is seen that the main clinical features of advanced cases of the disease were well known, although little or no distinction was made between tuberculosis of the lung and other chronic

wasting diseases of these organs. All sorts of conditions, as empyema, abscess of lung, tumour, gangrene, and bronchiectasis, were included under the term Phthisis. The association of chronic lung disease with clubbing of the fingers was well known, as the term "Hippocratic Fingers," which is applied to them, indicates. This clubbing is best seen in bronchiectatic conditions and not in tuberculosis. The disease was attributed by the ancients to a variety of causes, such as suppuration of the pleura, certain forms of pneumonia in which there was no crisis, blood spitting, and phlegm dropping down from the brain into the lung. This latter may simply have meant naso-pharyngeal mucus.

As far as can be discovered, little advance was made in the study of the disease for centuries. The Hippocratic conception of disease, as being due to a lack of proper balance between the four cardinal humours, blood, phlegm, yellow bile, and black bile, held captive the minds of men. These old opinions appear curious now to us with our more recent advances in medical science, but the profession has never at any time been backward in producing theories and of accepting them as facts, if the author of the theory has only attained sufficient eminence. In connection with tuberculosis, for example, even as late as 1875, we find a German savant giving expression to such fantastic ideas as that "tuberculosis in man depends on scrofula. This latter secretes a poison which when absorbed gives rise to tuberculosis." Another states that "the great danger in scrofula lies in the possibility of the supervention of tuberculosis." Statements such as those should lead us not to ridicule overmuch the condition of mind which produced the theories of the ancients, as even this generation may not be altogether guiltless.

The next writer, after Hippocrates, who interests us is Galen (130-200 A.D.). He appeared to recognise the infectious character of phthisis, but beyond that made no further advances. His idea of the pathology of the disease was that phthisis was caused by an ulceration of the lung. From this period down through the long sterile Middle Ages no progress whatever was made until the seventeenth century. The traditions of antiquity and a respect for

authority paralysed the intellectual activity of seekers after medical truth. The Renaissance, however, at last spread to medicine, and the "dead hand" of ancient dogmas and theories was cast aside. Henceforth men began to observe and note facts. Sylvius (1614-1672), a Hanoverian physician, wrote in his *Practice of Medicine*, when giving a description of phthisis, that the main symptoms were wasting, cough, spit, and fever. He added a contribution to the nomenclature of the disease by describing small swellings, which he called tubercles, in the lungs of those who had died of the disease. These tubercles, Sylvius thought, were glands in the lung which had become enlarged by disease, and which ultimately might soften and form cavities. Thus to Sylvius we are indebted for the name tubercle, the forerunner of the term tuberculosis.

Very little new ground was broken until the eighteenth century was well advanced, and by that time men had been taught to observe and to discard abstruse theories. It was then supposed that the tuberculous nodules in the lung were the result of some inflammatory process in the glands of the lung, but Baillie in the beginning of the eighteenth century showed that the tubercles arose in the cellular tissue of the lungs and that no glands existed in the lung tissue proper. In the year 1810 Bayle published his *Recherche sur la phthisie pulmonaire*, in which he gives an excellent description of the disease, and of the changes produced in the various tissues of the body by tuberculosis. He was the first to introduce the term "miliary" in connection with the small tubercles in the lung, and his term is still in use.

About this time also, Laennec (1781-1826) began his epoch-making clinical studies, and made the clear definite statement as to the unity of tuberculosis. He stated boldly that the caseous appearance and consistence of a tuberculous nodule in a lung or a gland was but a further development of the grey translucent appearance of an earlier deposit, and that there was no more difference between them than between the unripe and ripe stages of a fruit. "Tuberculous excavations," he further stated, "were the result of the softening and breaking down of these caseous nodules, and

the hæmoptysis which occurs so frequently in this illness is the result, rather than the cause, of the disease process." Just after Laennec's death the position was again much confused by the pathologist Virchow. This observer, who had established a world-renowned fame as a pathologist, introduced much perplexity into the subject by declaring that tubercles were not specific, that is, they could be caused by other diseases than tuberculosis. Virchow's opinions were readily and quite naturally accepted by his countrymen, and so far did some of them wander from the truth that another of these pathologists, Niemeyer, in 1866, declared that "the greatest danger a phthisical patient runs is that of becoming tuberculous."

At this very time, however, when the German medical men were wandering in the dark, one of Laennec's fellow countrymen, Villemin, demonstrated conclusively by animal experiment that tuberculosis is a single disease, thus confirming the statement of the great master, Laennec himself, when he spoke about the unity of tuberculosis. When Villemin inoculated various animals with tuberculous sputum or caseous material, he found that they all developed tuberculosis. He was unable, however, to demonstrate what the virus was. It was left for Koch in 1882 to establish, apparently for all time, the essential cause and unity of tuberculosis by the discovery of the Tubercle Bacillus. He demonstrated the bacillus in the diseased tissues, grew the organism on culture media, and reproduced the disease by injecting the culture into animals. In this way Koch gave signal and final proof of Laennec's dictum and demonstrated the existence of the causative organism that Villemin had assumed to be present.

In regard to the treatment, there have been alterations in the therapeutic outlook quite as revolutionary as those in connection with the causation of the disease. In the Middle Ages, phthisis shared, with all other diseases, treatment by weird and often repulsive prescriptions which were then in vogue. Here, for example, is a choice specimen from the seventeenth-century pharmacopeia of Willis which is taken from Miller's article on the famous old-time anatomist:—"Take of water, of snails, of earthworms, of

each an ounce and a half: of Liquid Laudanum Tartarized two drams: syrup of violets one ounce. Dose, one spoonful at bedtime." Or again, "Millepedes or Hoglice two drams; powdered seeds of nettles and burdock of each half a dram. Oil of nutmegs distilled one scruple: salt of amber half a dram. Juice of Liquorice Q.S. Make into small pills. Take three morning and evening."

Whether it was to cure a deadly disease, or to produce a love potion, the ingredients were much alike, a nauseous draught truly—frogs, marrow of a wolf's left foot mixed with ambergris, pigeon's liver, etc., etc. The addition of hippomane oftentimes had fatal results. The poet Lucretius and the notorious Caligula are alike said to have died from partaking of such prescriptions. By and by these medicines came into disrepute. Burton, the author of the *Anatomy of Melancholy*, was very sceptical of "apothecaries' physic" and urged people to stick to the Old Wives' "Kitchen physic," the "simples" of the delightful old-world herb gardens. Burton was careful, however, to add that Hippocrates (and others) in the infancy of their art were content with ordinary simples. Gradually these fantastic nostrums were turned to ridicule. Beaumont and Fletcher, for example, in one of their plays, *The Knight of the Burning Pestle*, has occasion to refer to a boy who complained that his feet were full of chilblains caused by travelling. His mother was thus advised:—

"Mistress Merrythought, when your youth comes home, let him rub all the soles of his feet and his heels and his ancles with a mouse-skin; or, if none of you can catch a mouse, when he goes to bed let him roll his feet in the warm embers, and I warrant you he shall be well." It was left to the Church of Rome to give a "serious account," Sir Thomas Browne tells us, "of a consumptive, hectic, phthisical woman, who was suddenly cured by the intercession of Ignatius." It should be added that among the sovereign remedies in the spacious days of Elizabeth were bleeding and purging. None was more popular than antimony in large doses, but there were hundreds of other remedies, which, as Burton puts it, "every gentlewoman in the country knows how to give."



These all had their day, but little advance was made until well towards the end of last century. Prior to the "open-air" regime it had been the fashion to try voyages to warmer climes, such as Madeira, where it was known that good results had in some cases been achieved. The medical mind, however, made the mistake of assuming that it was the warm air that was beneficial. Probably as a consequence of this, invalids at home were cooped up in rooms heated up to at least 70° F. into which no fresh air was permitted to enter. In 1840 Bodington wrote protesting against the methods then in vogue and advocated an open-air life, an idea that was taken up in Germany by Brehmer and his pupils. From this gradual development in the treatment of pulmonary tuberculosis has sprung up our present sanatorium system.

The outlook towards the disease has also undergone a revolution. Formerly it was thought that a diagnosis of consumption was tantamount to signing a death warrant, and doubtless this was correct in the great majority of cases, as it was only those in the later stages of the disease who were so classified. We now know that tuberculosis is a curable disease, and although the death-rate from it is still heavy, yet for every one who dies from it, ten recover. Thus, with the advance of medical science silently working its way down through the centuries, the darkness of years of superstition, of "amulets, spells, sigils, and incantations," has now given place to the light of common day.



## CHAPTER II

### EPIDEMIOLOGY

PULMONARY Tuberculosis is met with in all parts of the habitable globe. It is found in the Tropics, in the temperate zones, and in the Arctic regions. Amongst some of the uncivilised tribes of mankind tuberculosis did not exist until the advent of white traders. These brought with them some of the advantages of civilisation, but, as an off-set to the good things, they also introduced the curse of tuberculosis. Now tuberculosis is pandemic. Wherever vital statistics are kept, tuberculosis is always shown as contributing largely to the death-rate. This is revealed in the following table showing the mortality rate per 100,000 for the year 1912:—

France . . . . .	180	England and Wales . . . . .	104
Germany . . . . .	129	United States . . . . .	82
Austria . . . . .	283	Australia . . . . .	67
Italy . . . . .	107	New Zealand . . . . .	54
Spain . . . . .	120		

It will be seen that all countries do not suffer equally, some being considerably worse than others.

On closer examination it is found that in every country the distribution of the disease is very unequal. Generally speaking it is least in the country districts, more pronounced in the smaller towns, and most marked in the largest towns. Take the phthisis rate in Scotland for the year 1921 as illustrative, and we find that in

Rural Districts it is	68	per 100,000
Small Burghs	„	70 „
Large Burghs	„	95 „

Again, if the phthisis death-rate of any city is carefully analysed, it will be seen that the distribution of the

disease is still very unequal. Those parts of the city that are overcrowded and where poverty abounds always show an excess in the phthisis death-rate.

CITY OF EDINBURGH, 1921.			
Poorer Wards.		Better Class Wards.	
	per 100,000		per 100,000
Canongate . .	128	Morningside . .	70
St Giles . .	152	Newington . .	28
St Leonard's .	127	Haymarket . .	44

In this city, which may be taken as a type, the rates vary in different wards from 28 to 152 per 100,000.

A correct idea of the ravages of tuberculosis can only be had by taking the figures for the whole country and including all forms of the disease. In 1918, 611,861 persons died in England and Wales from all causes, and of this number 58,073 died of tuberculosis. In the quinquennium 1910-15, the enormous number of 254,194 persons died from all forms of tuberculosis.

Of all the various forms of the disease, pulmonary tuberculosis is the most important. Take, for instance, the death returns in 1913 for England and Wales, and it is seen that:—

49,476 persons died of tuberculosis (all forms).

Of these deaths, 37,055 were due to the pulmonary form,

5,018	„	tuberculous meningitis,
3,760	„	abdominal tuberculosis,
3,643	„	all other forms.

In other words, 76 per cent. of the total deaths due to tuberculosis were caused by the pulmonary form.

These figures show us something of the extent of the problem with which we are grappling, and demonstrate that tuberculosis is one of the largest problems, if not *the* largest single problem, with which Health Authorities have to deal.

# DEATHS FROM TUBERCULOSIS

9

## SCOTLAND.

DEATHS FROM TUBERCULOUS DISEASE, 1920.												
	All Ages.	Under 1 Year.	1-5.	5-10.	10-15.	15-25.	25-35.	35-45.	45-55.	55-65.	65-75.	75 and over.
Pulmonary Tuberculosis	4194	18	78	72	141	1016	976	813	631	308	117	24
Tuberculous Meningitis	590	146	218	102	60	41	13	6	4	...	...	...
Abdominal Tuberculosis	525	68	149	72	50	76	38	25	22	18	6	1
Other Tuberculous Diseases	733	51	93	63	61	146	94	76	61	55	21	12

DEATH-RATE PER 100,000 OF POPULATION.												
Pulmonary Tuberculosis	86	16	18	14	28	113	129	133	138	102	62	32
Tuberculous Meningitis	12	132	50	19	12	5	2	1	1	...	...	...
Abdominal Tuberculosis	11	61	34	14	10	8	5	4	5	6	3	1
Other Tuberculous Diseases	15	46	21	12	12	16	12	12	13	18	11	16

Taking the death-rate figures from another point of view, we find that tuberculosis causes 9.4 per cent., or roughly 10 per cent. of the total deaths. In other words, of *every ten persons born, one is going to die of this disease.*

**Age and Sex Distribution.**—A study of the age and sex distribution of the deaths from tuberculosis shows that the disease does not fall equally at all ages, nor are the deaths equally distributed between the sexes.

If we study the age distribution as shown on chart (Fig. 1), we find that in the first year of life there is a heavy death-rate, nearly 288 per 100,000, from tuberculosis, but chiefly due to the non-pulmonary type. Between 5 and 15 there are fewer deaths from tuberculosis than at any

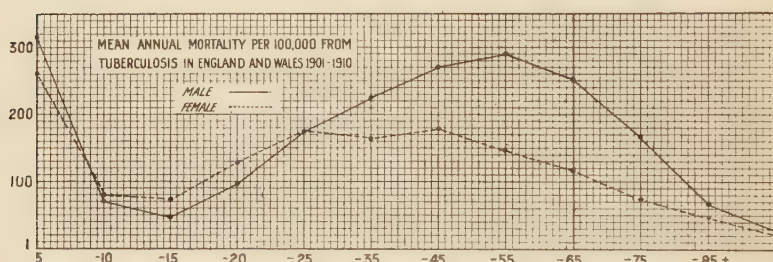
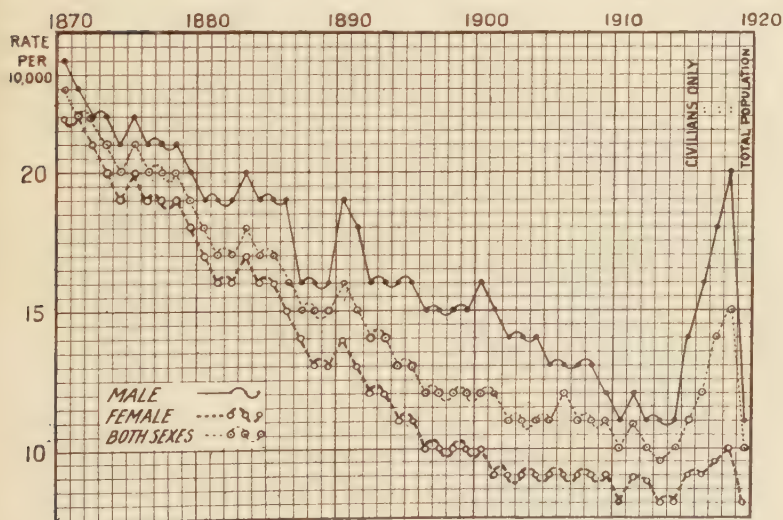
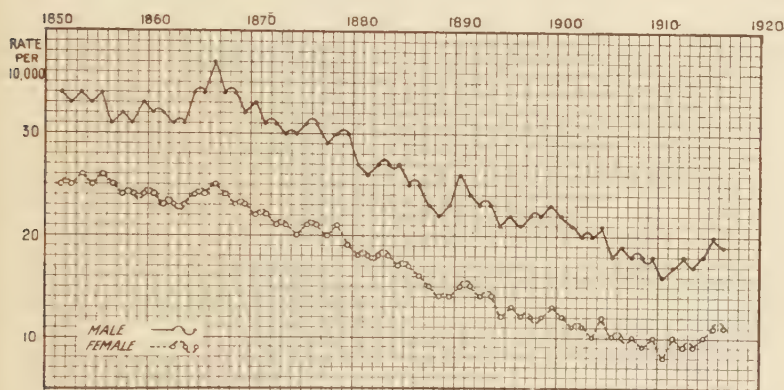


FIG. 1.

other period in life. After 15 the mortality rate rises rapidly, and the pulmonary form of the disease contributes by far the greater number of deaths. After 15 nearly 90 per cent. of the deaths from tuberculosis are due to it. The maximum death-rate is reached between 35 and 55 years. After this the rate diminishes rapidly. There are thus two periods of life when tuberculosis contributes most prominently to the death-rates. The first is under 5 years; the second is from 35 to 55 years. With regard to the first period, viz. from birth to 5 years, nearly one-half of the deaths occur under 1 year.

It is an elementary truth to those who have studied this disease that more men die of tuberculosis than women. This disparity is more marked in urban communities than in rural. The difference is well shown in the case of London, where the male and female rates are shown in the graph (Fig. 2).

This difference between the sexes did not always exist. In England and Wales before about the year 1865 more women than men died from the disease, but after this period



the death-rate amongst women has been less. The difference in succeeding years between the sexes is shown by the graph given above (Fig. 3).

The ratio of the male to the female deaths at different ages is of interest. Taking the female as 100, the following are the comparative figures for the males in England and Wales from 1901-1910:—

Age.	Males.	Age.	Males.
5 . . . . .	118·7	55 . . . . .	202·5
10 . . . . .	91·1	65 . . . . .	217·0
15 . . . . .	65·2	75 . . . . .	188·8
20 . . . . .	79·8	85 . . . . .	122·8
25 . . . . .	122·4	Over 85 . . . . .	132·8
35 . . . . .	130·8	All ages . . . . .	133·8
45 . . . . .	153·3		

This difference between the sexes is of much interest. Between 5 and 20 the females contribute a greater number of deaths, but after 30 the proportion of males steadily increases, and this increase is maintained during the remainder of the age periods. In the age period 45 to 65 the male deaths are more than double those of the females. The explanation of the difference in the sex rate is to be found, probably, in the occupations of the sexes. In our country the majority of the female population is occupied with home work, whereas the men are industrialised and spend their days in factories and workshops which in many cases are inadequately ventilated.

In cities like Dundee, where a large proportion of women work in the factories, the difference between the male and female death-rate is less marked.

Professor Hunter Stewart shows that in the years 1901-1907 the mortality rate in Dundee was as follows:—

Male tuberculosis death-rate . . . . .	175 per 100,000
Female       "       "       " . . . . .	170       "

As tending to confirm this view, it should be pointed out that during the Great War, when a greater proportion of the female population was called upon to enter industrial life, the death-rate from tuberculosis increased more amongst the females than the males.

The increase of deaths amongst the female population during war time should not, however, be attributed solely to their industrialism. It must be remembered that the whole nation was strictly rationed and that food-stuffs were not to



be had in their former abundance or variety, and it is a well-known fact that when food becomes scarce in a household it is the mother who first denies herself that the others may benefit. While it is impossible to give figures showing the effect of this factor upon the female death-rate, it is admitted that it is an important one and must be kept in view.

DEATHS FROM PULMONARY TUBERCLE.							
	Male.	Female.	Increase above 1913.				
			Male.	Per cent.	Female.	Per cent.	
1912	27,801	22,243					
1913	27,649	21,815					
1914	28,071	22,214	422	= 1·5	399	= 1·8	
1915	30,332	23,940	2683	= 9·7	2125	= 9·7	
1916	29,724	24,131	2075	= 7·5	2316	= 10·6	
1917	30,357	25,574	2708	= 9·8	3759	= 17·2	
1918	31,025	27,046	3376	= 12·2	5231	= 23·9	
1919	24,549	21,761	Dec. 2100		Dec. 54		

**Extent of the Disease.**—It is difficult to arrive at a correct estimate of the actual extent of the disease from the death-rates, but it has been commonly assumed that for each death from the pulmonary form of the disease there are ten who are suffering from it. Such figures as are available go to support this assumption.

In the City of Edinburgh, in the year 1920, there were recorded 287 deaths from pulmonary tuberculosis, and taking the proportion of ten sufferers to one death, that would show 2870 patients actually suffering from that form of the disease in any one year.

From the register of notifications it was found that there were actually 2000 known cases of pulmonary tuberculosis in the city at that time. This is a minimum figure, as a considerable number, say 500 to 700, were then ill but not yet notified. Taking the round figure of 2700 as representing approximately the actual cases in the city, this shows a close agreement with the ratio of about ten sufferers to every death. Accepting this figure for the purposes of the argument as correct, this would show that in England and Wales in 1918 there were over 450,000 patients suffering from pulmonary tuberculosis alone.



To this number must be added those cases of tuberculosis other than pulmonary. To get the number of these is even more difficult, but it is safe to assume that it is approximately one-quarter of that for pulmonary tuberculosis. This will give a total number of well over half a million of sufferers, which may be taken as an extremely conservative estimate, and yet it presents some idea of the extent of tuberculosis in our midst.

**Prevalence of Infection.**—There is another aspect of the problem which must now be discussed, viz., the prevalence of infection, and in order to get a correct idea of this we must distinguish between those suffering from active tuberculosis and those who are merely infected with the germ. This distinction must be kept in mind, as many are infected who do not suffer from active tuberculosis. There are two sources from which we derive our knowledge of the extent of tuberculous infection, viz.—

- (1) From pathological findings, and (2) from the tuberculin tests.

(1) **Pathological Findings.**—It has long been observed at post-mortem examinations that frequently there was evidence of tuberculous lesions, although the persons had died from non-tuberculous diseases. Many of these lesions were found to be in a healed condition. Nageli's figures are well known. Out of 500 post-mortem examinations he found that 71 per cent. showed pathological changes due to tuberculosis. In those whose ages were over 18, no fewer than 98 per cent. were infected, although only 28 per cent. of them had died of the disease. In the other 70 per cent. of the cases the disease was quiescent or only slightly active. In the cases under 18 years of age, only 25 per cent. showed evidence of tuberculous infection, and the lesions, as a rule, were in an actively progressive condition. Other observers have produced further evidence. Opie in fifty autopsies, in persons of varying ages from 18 upwards, found that all of the cases showed evidence of tuberculosis, but that in only four of the cases was the disease fatal.

In another series of ninety-three autopsies under 18 the distribution is as follows:—

Age.	No. of Autopsies.	Tuberculosis present.	Fatal.
		Per cent.	
1 year . . .	43	4 = 9.3	4
1- 2 years . . .	16	1 = 6.2	1
2- 5 „ . . .	14	6 = 42.8	3
5-10 „ . . .	11	5 = 45.5	2
10-18 „ . . .	9	6 = 66.7	1

Hamburger gives the following table, the result of 848 autopsies on children:—

Age.	Number.	Tuberculosis present.
		Per cent.
3 months . . .	105	4 = 4
3- 6 „ . . .	73	13 = 18
6-12 „ . . .	140	32 = 23
1- 2 years . . .	179	74 = 40
2- 4 „ . . .	175	102 = 60
4- 6 „ . . .	67	38 = 56
6-10 „ . . .	65	41 = 63
10-14 „ . . .	44	31 = 70
	848	335 = 40

From a study of the findings of the pathologists two facts emerge:—

- (a) The infection increases rapidly as childhood advances, until practically all of us become tuberculised in early adult life.
- (b) The younger the child is infected, the greater the chance of the disease being progressive.

It will be noted that in Opie's figures, out of forty-three autopsies under the age of 1 in the four cases where evidence of tuberculosis was found, the infants had all died of the disease.

As far as the tuberculous infection of civilised man is concerned, the pathologist proves that the great majority of adults have been infected at one time or other.

(2) **Tuberculin Tests.**—The delicacy and the specificity of the cutaneous tuberculin reaction being now recognised and accepted, this test furnishes us with valuable information regarding the widespread character of tuberculous infection.

Von Pirquet summarises the result of 988 cases as follows :—

Age.	Positive Reaction. Per cent.	Age.	Positive Reaction. Per cent.
0- 3 months . . .	0	4- 6 years . . .	53
3- 6 „ . . .	5	6-10 „ . . .	57
6-12 „ . . .	16	10-14 „ . . .	68
1- 2 years . . .	24	Over 14 years . . .	90
2- 4 „ . . .	37		

Although some other observers have failed to get as heavy an incidence, yet these figures may be taken as approximately correct.

Calmette gives the following table as a result of the test on 1226 children :—

Age.	No. of Children.	Positive Reaction.
		Per cent.
0- 1 year . . .	273	8·7
1- 2 years . . .	145	22·1
2- 5 „ . . .	206	55·8
5-15 „ . . .	366	81·4
Over 15 years . . .	236	87·7

We have already seen that the pathologists show that the great majority of us have at some time been infected by tubercle, and now the clinicians demonstrate that approximately 90 per cent. of us after the fourteenth year are infected. We may thus take it as proven that practically all of us have had at one time or other in our history to resist an attack by the tubercle bacillus. A most encouraging fact, however, is that although all of us are infected, yet only 10 per cent. of us die of the disease. The remainder recover.

**Fall in the Death-Rate.**—The behaviour of tuberculosis in its social aspect has attracted much attention, as for many years past the numbers of deaths due to it have been steadily declining. This has been so marked that there is

every hope that should the fall continue in the future at the rate shown in the past, the disease in another twenty-five years will be wellnigh wiped out of the mortality tables.

The fall in the death-rate from pulmonary tuberculosis is full of interest and encouragement. In examining the graph (Fig. 4) which represents the Scottish death-rate, it will be seen that it runs at a high level and without any definite tendency to fall until after the year 1870. In that year it

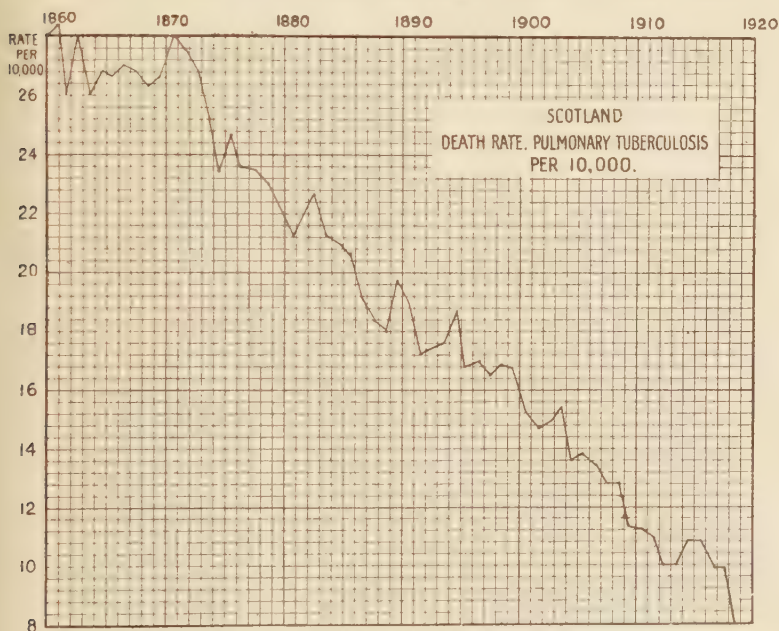


FIG. 4.

reached its highest point, and then from some cause or causes it began to decline, and the fall has been continuous up to the present. In 1870 the death-rate per 100,000 in Scotland from pulmonary tuberculosis was 280, and in the year 1919 it was 88. This represents an enormous saving in human life. During the year 1919, 4294 persons died in Scotland of pulmonary tuberculosis, but if the rate which prevailed in 1870 had continued in 1919 the number would have been approximately 13,661. Therefore in that year about 9367 lives had been preserved. In England and Wales the

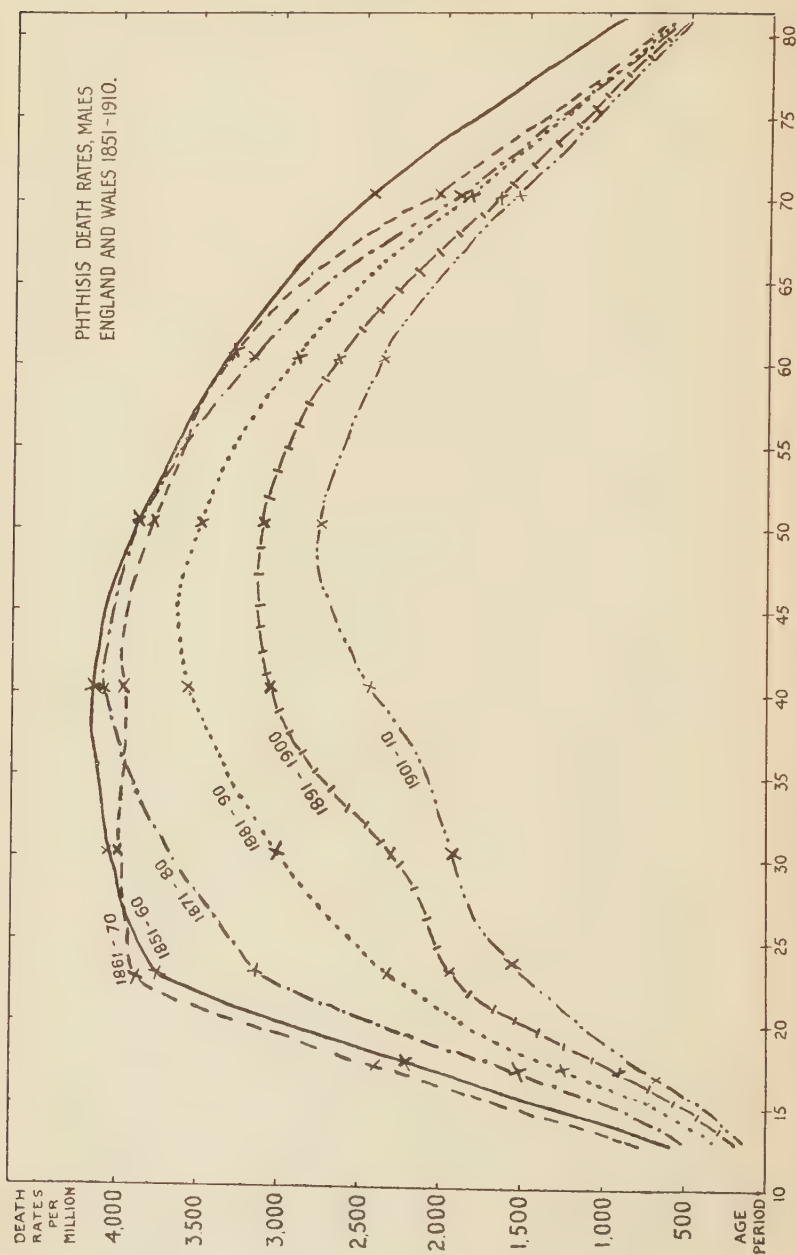


Fig. 5.

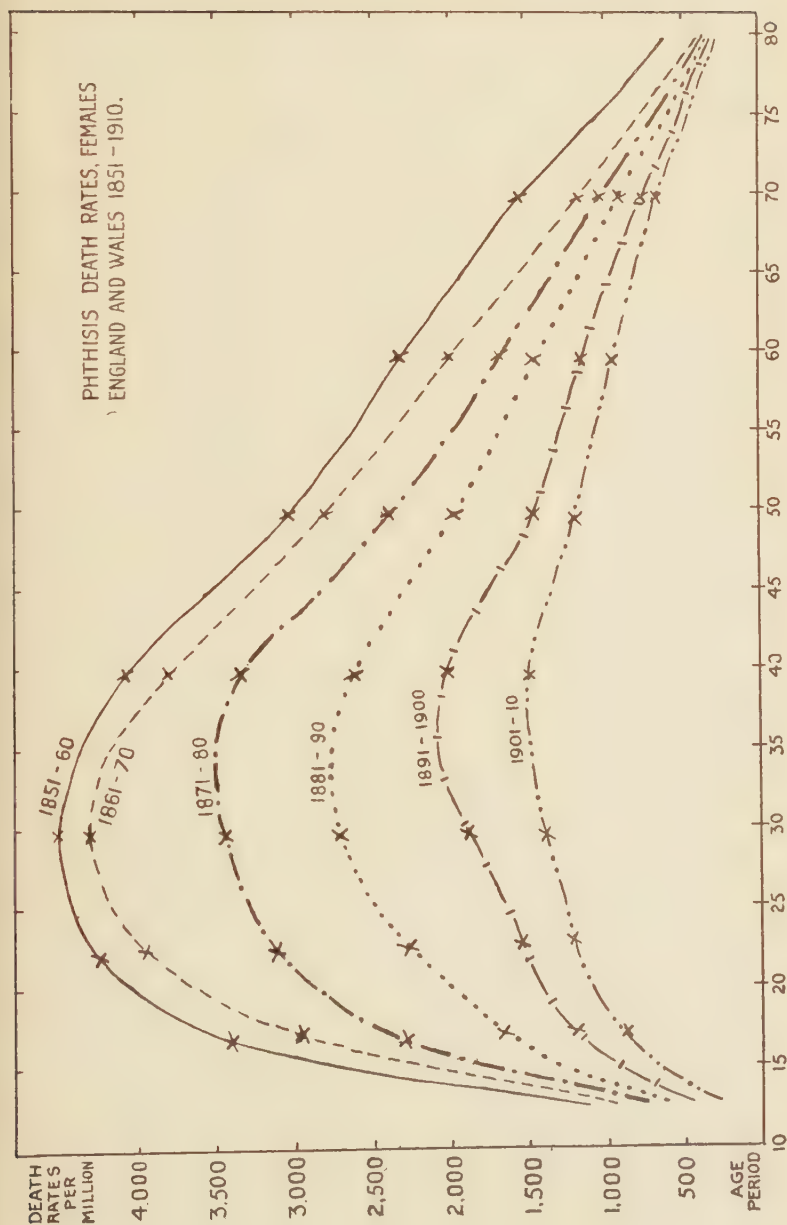


FIG. 6.



death-rate from pulmonary tuberculosis shows a similar fall. This is well seen in the above charts (Figs. 5 and 6), taken from Dr Brownlee's report to the Medical Research Committee, which shows the fall in males and females respectively.

#### DEATH-RATES FROM PULMONARY TUBERCULOSIS.

Deaths per 100,000.						
	1881 to 1885.	1886 to 1890.	1891 to 1895.	1896 to 1900.	1900 to 1905.	1906 to 1910.
Vienna . . .	685	576	474	381	336	274
Paris . . .	441	440	409	379	390	374
Moscow . . .	411	393	391	324	268	258
Boston . . .	411	377	289	240	217	175
New York . . .	398	350	286	242	215	197
Munich . . .	389	348	312	303	269	226
Dublin . . .	346	341	335	317	309	268
Glasgow . . .	311	250	227	195	170	140
London . . .	222	197	185	175	157	114
Edinburgh . . .	212	191	180	187	157	114
Sydney . . .	193	157	119	98	98	72

In every town there is a marked fall, and these towns are taken from the Continent of Europe, North America, and Australia.

A marked exception to this world-wide fall is in Ireland. Here the curve has little tendency to fall until the year 1900. There has been a good deal of doubt expressed as to the accuracy of the Irish curve, and the explanation offered is that in former years diagnosis was very loosely made, and in later years, when greater care was exercised, the number of deaths from this disease apparently increased. With this one noteworthy exception, however, the apparent fall in the death-rate from pulmonary tuberculosis is universal. This very striking feature is worthy of more detailed study. There are two main points which should be discussed in connection with the graph illustrating the fall in the phthisis death-rate:—(1) The accuracy of the curve; (2) the causes affecting the curve.

(1) In considering the accuracy of the curve, it has to be remembered that the diagnosis of advanced pulmonary



tuberculosis is comparatively easy. There is nothing like the same room for doubt as there is in the diagnosis of early tuberculosis. This being so, it is safe to assume that in the past thirty years the death certification has been fairly accurate. During the previous twenty years there may have been mistakes in diagnosis, but not so many as to alter the curve materially. There can be no doubt, however, that in the earlier period referred to many of the rarer diseases which produce wasting prior to death would be undoubtedly classed as "phthisis." This, of course, would tend to raise the rate for pulmonary tuberculosis during that period. Cancer is another disease which might affect the death-rate in this fashion. It is well known that there has been a marked rise in the death-rate from this disease during the period in which the tuberculosis death-rate has been falling. The increase in the cancer death-rate is due chiefly to the diagnosis of "internal" cancer; and there is little room for doubt that in the earlier days many of these cases would, through wrong diagnosis, be entered under, and thus raise, the tuberculosis death-rate. On the other hand it is likely that many cases of tuberculosis, especially of the more chronic types, would be certified as bronchitis, and this would tend to lower the rate for tuberculosis.

Taking a comprehensive view of the curve, it can be accepted that for the past thirty years it is quite reliable, but in respect to the previous twenty years there may be some question as to its exactness. The errors on the one side, however, are probably balanced by the errors on the other, and the curve may be taken as approximately correct. The fall in the death-rate from pulmonary tuberculosis is thus not apparent only, but real.

Accepting, then, the curve as representing actual facts, the causes which have been suggested as contributory to this fall should now be examined.

(2) These causative factors can be grouped under the following heads:—(a) Biologic; (b) Sanitary; (c) Economic; (d) Educational.

(a) **Biologic Factors.**—It has been suggested that the fall in the death-rate is due to a change in the type of disease, that its virulence is lessened, hence fatal cases are

fewer. To support this contention, an analogy is drawn from scarlet fever. In this disease it is well known that the mortality rate is much lower now than it was formerly, and this lessened mortality rate is assumed to be due to a change in the type of disease—the result of a diminished virulence of the bacillus. It is possible that this assumption may not be correct, and the diminution in the death-rate may have been brought about by better nursing facilities and improved hospital accommodation, with isolation of the more virulent cases and increased resistance. Hence it would appear that the suggestion that the decline in the phthisis death-rate is due to a lessened virulence of the bacillus is based on a somewhat shaky foundation.

Another theory which has been advanced to explain the decline is Cyclic Periodicity. Certain diseases come in cycles. For a time there are few cases with us, and then there comes a wave of the disease which reaches its maximum, and then ebbs again. Measles is a typical example. Every two or three years widespread epidemics sweep over the land; these pass, and the disease subsides to the average. It has been argued that, in like manner, pulmonary tuberculosis comes in great tides, the ebb and flow occupying scores of years, and that at present we are at ebb-tide. This is ingenious, but not very convincing, and there appears to be no proof of the assertion.

Yet another theory demands examination. Its supporters state that the reduction of the phthisis death-rate is in great part due to acquired immunity. As the generations succeed each other, where pulmonary tuberculosis abounds, each successive generation acquires an increased resistance to the disease as the susceptible are killed off. There can be no doubt that the inhabitants of Britain have a much greater power of resistance to the tubercle bacillus than the coloured natives of South Africa, and it is reasonable to assume that this is because of the prevalence of the disease amongst us for countless generations. There may be some truth in the theory of acquired immunity but it is impossible to appraise its amount or influence. The experience of Ireland would, at first sight, appear to throw doubt on it, as the death-rate in that country rose from 1.7 per 1000 in 1864

to 2.2 per 1000 in 1900. If these figures represent the facts, here we have a people and civilisation akin to that of Britain, and yet the disease is not producing immunity. Ireland, however, presents special features which may account for this apparent increase, such as the Irish Famine of last century and the subsequent emigration of the young and able-bodied from that country, but grave doubts have been thrown on the accuracy of the figures.

Cobbett introduces a new idea in regard to the decline, viz. the immunisation of the race by the small frequent doses of bacilli which all of us who are city dwellers receive. Carrying this theory of immunisation further, he states that if it is playing any important part in increasing the resistance of the present generation, it is just possible that by checking the distribution of the bacilli by discouraging indiscriminate spitting, and by abolishing bovine tuberculosis from dairy cattle, we may be actually undermining the powers of resistance of the race and paving the way for a future increase in the severity of the disease. Few of us, however, are likely knowingly to inhale human bacilli or to swallow bovine bacilli on the grounds that they may prevent a severe or fatal attack of the disease at a future date.

(b) **Sanitary Factors.**—Prior to the last three-quarters of a century very little attempt was made towards bringing about a sanitary condition of towns or villages. Drainage and sewerage works were practically unknown, and in the poorer parts of many towns the refuse and excreta were thrown out into the street, to pollute the air and contaminate the soil, so that the inhabitants were exposed continually to their devitalising influences.

It is not to be wondered at, then, that under these circumstances the general death-rate was high, that waves of epidemics swept over the land, and that tuberculosis found easy victims in the individuals whose tissue resistance had been lowered and vitiated by these conditions. One of the earliest attempts that was made to improve the conditions of the towns was the appointing of a Commission of Health of towns in 1842. Such a lamentable condition of things was revealed in the report of this Commission, that several laws were enacted to remedy matters such as the Building and Ventilation Law in 1845, and the first Public Health Act in

1848. With the gradual growth of public opinion, and with the experience of the working of these Acts, the way was paved for the great Public Health Act of 1875. These Acts dealt with sanitation in its widest sense; they took care of the purity of the water supplies, insisted on the drainage and paving of the streets, and on the supervision of nuisances and uncleanness generally. With regard to the drainage of the streets and subsoil, it is stated in the ninth report to the Privy Council that this has led to the diminution, more or less considerable, of phthisis. Buchanan and Bowditch have also produced figures which appear to corroborate this statement, and though later observers have questioned the accuracy of the deductions of these individuals, there is no doubt that the improved sanitation of the towns has had profound effects upon the health of the citizens.

One factor which must not be overlooked here is the repeal of the window tax in 1851. From that date, in all the newer houses, window space would be looked upon as a desirable thing, instead of being regarded, as formerly, as a thing to be avoided as far as possible. The influence of sunshine on bacteria is too well known to require further elaboration. Little is heard, nor indeed can anything be directly proved, as to the effect of the repeal of the window tax, but doubtless its results were far-reaching.

*Housing Acts.*—Several Acts were introduced to ameliorate the housing conditions of the cities, and under their influence large areas of slum properties have been dealt with and removed. There can be no doubt that the improvement brought about in the housing conditions of the city populations has been a great factor in the fall of the death-rate, as wherever slum conditions are found there the tuberculosis death-rate is highest. This is well brought out in numerous health reports from towns such as Edinburgh, Glasgow, and Greenock. The populations living in the 1, 2, 3, and 4 roomed houses are taken, and invariably the highest proportion of tuberculosis is to be found in the 1-roomed houses. In the population dwelling in 4, and more than 4, roomed houses, a much smaller death-rate from tuberculosis is found. It should be noted here, however, that other factors besides slum houses have a bearing on the incidence of tuberculosis; the character of the inmate, his social and economic position,



his educational and hygienic standards, and his environment have all to be considered, as each plays its separate part.

*Factory Acts.*—As the rural populations crowded into the industrial centres many nuisances arose in connection with factories and workshops, and in order to remedy this various Factory and Workshop Acts were passed. One of the main points to be considered as far as health is concerned, is that they insisted in the factory upon a certain cubic space per individual. The law requires a minimum of 250 cubic feet, or in the case of a man working overtime, of 400 cubic feet, whilst certain other provisions are made with regard to the cleanliness of the factory or workshop, and the removal of dust by the installation of powerful fans, etc. In the case of the cotton-spinning sheds, a beginning has been made by setting up a standard for the maximum amount of carbon dioxide permissible in the air—more than 0.09 per cent. is not permitted. This is a matter which ought to be insisted on in all workshops and factories, so that it would be obligatory to maintain a certain purity of atmosphere. Too often no attention whatever is paid to the actual condition of the atmosphere, so long as the provision of certain openings for ventilation exist. Unfortunately the working people are apt to keep these openings carefully closed, with the result that their effect is nullified. In order to obviate this, the ventilation of the workshops and factories should be so arranged that it would be impossible to interfere with the free and adequate ingress of fresh air.

(c) **Economic Factors.**—A survey of the various factors which have aided the reduction of the death-rate would be incomplete without note being taken of the improved economic conditions. The Repeal of the Corn Laws in 1846 was a step of prime importance, as it was followed by the importation of a gradually increasing amount of cheap food into this country.

It may not be altogether a coincidence that the Scottish death-rate from tuberculosis began to fall in 1870, *i.e.*, twenty-four years after the passing of the Repeal of the Corn Laws Act. It is to be presumed that the generation born after the passing of the Act would be better fed in infancy and adolescence than those born in the “hungry forties,” and consequently when they reached the ages at

which under the old conditions the death-rate from phthisis began to be heavy, the new generation with their improved feeding would resist the disease better than their predecessors, and this, naturally, would affect the death-rate. Too much stress should not be laid on this point, as no such corresponding effect is seen in the English rate.

Another point to be considered under this heading is the very marked increase in the general national prosperity since about 1870. Wages have increased, and there has been an abundance of cheap food. Better housing and better standards of nutrition and cleanliness prevail. The reduction of poverty is a most important factor, as death-rates are highest in the districts where poverty abounds; nor is this to be wondered at, as poverty implies insufficient feeding and insufficient clothing, poor housing in crowded localities, and too often dirt and ignorance. Insufficient food and clothes often imply ill-ventilated rooms, as poorly fed and ill-clad individuals cannot stand exposure to cold as the well-fed and well-clothed do, and in consequence they attempt to warm their rooms by preventing all ingress of the cold though fresh air. With improved economic conditions there comes a rise in the well-being of the home.

(*d*) **Educational factors** also count for much. We have now had several generations of compulsory education, and there is a growing enlightenment on the part of the general population in the elements of hygiene and the preservation of health. This knowledge, while not yet either deep or extensive, is not without its influence on the tuberculosis problem, but as it deepens and widens it will form a powerful factor in the final abolition of the disease. The nutritional, the educational, and the hygienic standards all rise, no doubt at varying rates, but nevertheless they rise.

Newsholme has made the interesting suggestion that the most important factor in the decline is the segregation of the advanced pauper consumptives in the workhouses—a suggestion, however, with which I personally am not inclined to agree.

No one factor explains the almost universal fall in the death-rate from this disease, but every step towards a higher standard of living, better housing, and improved hygiene generally is a step towards the abolition of phthisis.

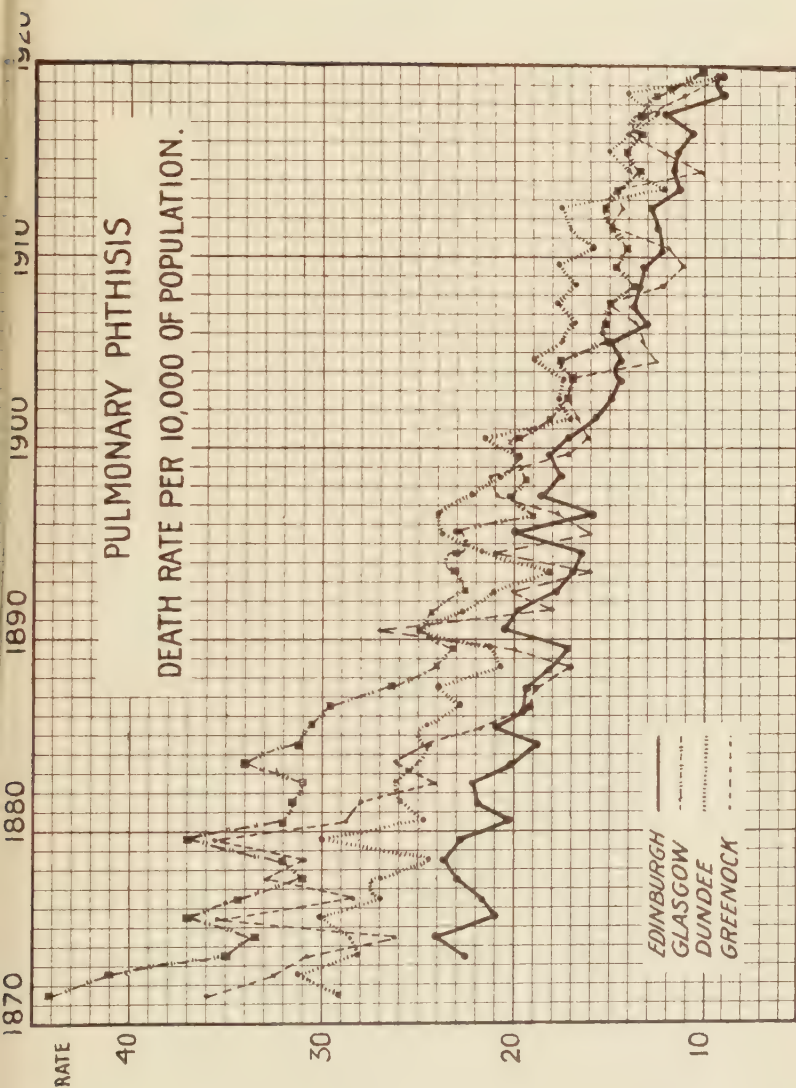


FIG. 6A.—Chart showing Fall in Various Towns.



## CHAPTER III

### THE TUBERCLE BACILLUS

THE tubercle bacillus was discovered by Koch in 1882. Prior to this some interesting and some grotesque theories as to the cause of tuberculosis had been advanced, but the discovery of the bacillus settled the question permanently. Its presence in all tuberculous lesions also settled permanently the unity of the disease, no matter how varied its manifestations might be.

**Morphology.**—The bacillus is a small slender rod which varies slightly in size. Usually its length approximates half the diameter of a red blood corpuscle, so that it measures from 2 to 4 micromillimetres in length, and about 0.4 in breadth. It is slightly curved, with well-rounded ends, and is non-motile. It consists of a central protoplasmic portion surrounded by a thin capsule of fatty or waxy material. There is no direct evidence to show that it forms spores. Some specimens, when stained, show a beaded appearance, with some portions more deeply stained than others, simulating spore formation; but other tests, such as those of their exposure to antiseptics, heat, or sunlight, show that they have not the resisting power which spores usually possess. The absence of direct evidence of spore formation does not, however, rule it entirely out of the question. Occasionally some bacilli are met with which show a tendency to branching or may even be Y-shaped.

The variation in length and staining properties are commonly assumed to have some definite clinical bearing. Short, uniformly well-stained bacilli, especially if found in clumps, are taken to mean that the disease in the lung is in a fairly active condition. On the other hand, long, curved, irregularly stained bacilli are associated with non-progressive

disease. Too much importance should not be attached to this, however. The bacillus is, as a rule, longer when found in sputum than when grown on an artificial medium. The germ is not found free in nature but always inhabiting some host, such as man.

**Growth.**—The bacillus is aërobic and can be cultivated on various artificial media. Its growth is extremely slow, and the temperature best suited for its growth is about 37° centigrade. It can grow, however, at temperatures of from 29° to 40°. At the end of three or four weeks, depending upon the medium on which it is grown, there is found a slight yellowish-white growth irregular at the margin and scaly on the surface. In old cultures the colour may become brownish. It grows well on glycerine bouillon, forming in a few weeks a thin fragile growth on the surface of the liquid, with a characteristic wrinkled appearance. Cultures may be obtained directly from a tuberculous nodule by taking a small freshly-cut portion of the nodule under strict aseptic precautions and smearing thoroughly the surface of a blood serum tube with the cut surface.

It is much more difficult to grow it directly from sputum, owing to the presence of other organisms whose rate of growth far exceeds that of the tubercle bacillus. It can, however, be done by the antiformin method. This consists in mixing the sputum with a certain percentage of antiformin, which kills off the organisms other than the tubercle bacillus. The following is the method adopted by Griffith. Mix the sputum with an equal volume of 10 per cent. antiformin, and stir until the sputum is dissolved. This takes from five to fifteen minutes according to the consistence of the sputum. The mixture is next centrifuged, the clear upper layer decanted off, and the deposit washed by mixing it with normal salt solution. This mixture is again centrifuged, and the deposit is inoculated on several tubes containing egg medium and incubated.

More recent investigations by Griffith have modified the above method considerably. He finds that for routine purposes it is better to use 15 per cent. antiformin, and after stirring the mixture, to inoculate the tubes without centrifugalising. At least three tubes should be inoculated in the

first five minutes, and the first two should be made one or two minutes after the mixture. A few more tubes should be inoculated at intervals of five and ten minutes respectively.

The usual method of cultivating the bacillus is by inoculating a guinea-pig with some tuberculous material. After three weeks or so the animal is killed. The infected glands are removed and a freshly cut portion of one smeared on a serum tube. The ordinary media on which it is grown are blood serum, Dorset's egg medium, or glycerine agar. On the latter the growth forms the characteristic white layer already spoken of. Secondary cultures grow more readily and more luxuriantly in glycerine agar. The bacillus grows readily on other media, such as sputum agar and lung agar.

**Power of Resistance.**—It has been estimated that a single consumptive will expectorate daily sputum containing many millions of tubercle bacilli. When it is remembered that many patients expectorate carelessly, it will be seen that the viability of the bacillus when expelled from the body is a matter of importance. It has been shown that direct sunlight is one of the best destructive agencies. Exposure of the bacilli to the direct rays of the sun invariably kills them. It is supposed to do so by direct destruction of the fatty envelope. The time necessary for this varies according to the medium in which the bacilli are. In a thin layer of sputum it has been found that the bacilli are killed in from a few minutes to a few hours. However, when in *fæces*, the time necessary amounted to several days. Diffuse daylight kills the germs but requires a much longer time. Some observers have noted that they were killed within twenty-four hours, but usually about one week was required. Other observers, however, state that the bacilli were alive after an exposure of about four months. In dark rooms they have been found alive and virulent for at least a year. In dried sputum the bacilli retain their virulence for periods up to six months. When desiccated they can resist dry heat up to 100° C. for an hour, but exposure to moist heat of 70° C. usually kills in about twenty minutes. In milk they are destroyed in fifteen to thirty minutes if the temperature is raised to 70° C. If the temperature be raised to 100° this is sufficient to kill them in three minutes. Freezing has

apparently little effect on the bacillus, and this fact should emphasise the importance of careful inspection of the enormous quantities of frozen meat imported into this country.

The bacilli are very resistant to the products of putrefaction. They can survive for many months in sour milk, and exist for a long period in putrefying sputum or sewage. They have been found in an active virulent condition in butter or cheese which has been made from milk containing tubercle bacilli. As a rule, in these latter substances they die out in a few weeks, but instances have been given when live bacilli have been found many months after these articles had been made up. In tuberculous meat they are usually killed by cooking, although in a large joint some of the germs in the centre may escape as the heat does not penetrate to the centre sufficiently to effect their destruction.

Chemical antiseptics vary in their destructive power over the tubercle bacillus. In sputum a 5 per cent. solution of carbolic kills with an exposure of not more than a few minutes, but mercuric chloride solution is of little value in tuberculous sputum as it coagulates the mucin and albumen, and this forms a protection to the bacillus. Lysol is a fairly rapid disinfectant, and it has the additional advantage that it acts as a solvent on the mucus of the sputum.

**Staining.**—Tubercle bacilli stain with difficulty. They must be treated for a prolonged period unless the action of the dye is hastened by heating. When once stained they are difficult to decolorise. They will resist the decolorising effect of fairly strong acid solutions. Hence the term applied to this and other somewhat similar organisms, “acid-fast.” The most common, and probably the best, stain is the Ziehl Neelsen carbol fuchsin stain. It is made up and used as follows:—

(a) Carbol fuchsin solution.

Basic fuchsin, 1 gram.

Carbolic acid crystals, 5 grams.

Absolute alcohol, 10 c.c.

Distilled water, 100 c.c.

- (b) Solution of sulphuric or hydrochloric acid, 25 per cent.
- (c) Absolute alcohol.
- (d) Watery solution of methylene blue.

*Procedure.*—A small opaque portion of sputum is picked up, and a somewhat thick film made with it on a glass slide. It is then fixed by passing two or three times through the flame of a Bunsen burner. The film is next covered by the carbol fuchsin solution and heated over the flame until it steams. It is allowed to remain covered with the hot solution for about five minutes. The surplus fluid is then poured off and the slide washed in running water. It is then bleached by dropping the acid solution on it. This may take a minute or two, and care should be taken not to overdo the bleaching, which is best avoided by alternately flooding the slide with the acid solution and water. The decolorising process should be stopped while there is still a faint pink colour in the film. It should be further treated by washing in alcohol until no more red colour can be washed off. This usually takes from a half minute to one minute. It is then quickly washed in water and counterstained with the methylene blue solution for a minute or two. It is again washed in water, dried, and is now ready for examination. This is probably the best stain we have, as it is one of the oldest. There are many other modifications, but it is unnecessary to deal with them here.

In some tuberculous lesions there are found bacilli or parts of bacilli not affected by Ziehl Neelsen stain, but capable of staining by Gram's method. These have been called Much's Granules after the observer who first described them. They are usually found in arrested or fibroid lesions, cold abscesses, etc. Because of this they are supposed to be degenerated forms. These granules, however, are still virulent, and under suitable conditions will grow and develop like the ordinary bacillus.

**Distribution.**—Seeing that the bacillus lives and multiplies only in the animal organism, it is of interest to note how it becomes distributed outside the body.

*Sputum.*—So far as the human type of the bacillus is

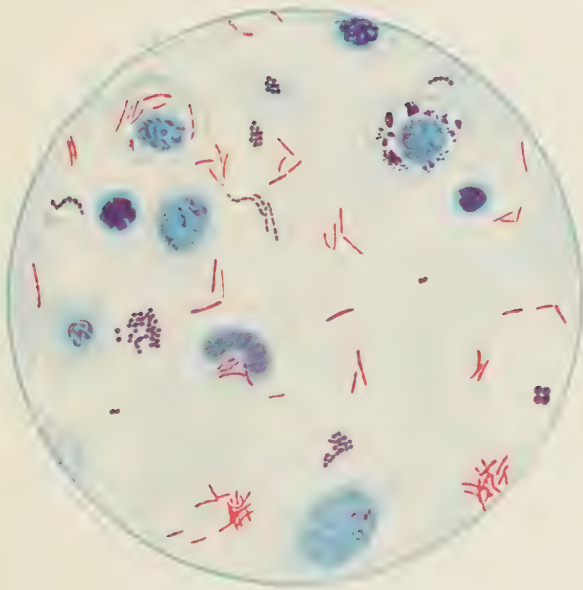


FIG. 7.—Sputum containing Tubercle Bacilli.  $\times 1000$  diam.





concerned, spitting by the consumptive is the common method of distribution, but the extent of this is greatly influenced by the habits of the infected person. The patient who is careless with his sputum is a source of danger to those around him. Virulent bacilli have been demonstrated on the hands of such persons, on their clothing, and on their bed linen. Their feeding utensils and the dust of their rooms have been shown to be contaminated. Even on the hands of the attendants of such careless individuals virulent bacilli have been demonstrated. Where the consumptive is careful as to the disposal of his sputum, the distribution of the bacilli is much more limited. Thus a well-trained consumptive may live in a house and be of comparatively little danger to the other inhabitants. In a well-conducted sanatorium the dust in the rooms should be free from tubercle bacilli. These facts indicate the necessity for careful training of all known cases of consumption, and in this direction the numerous sanatoria which we have in this country may bestow enormous benefits upon the community.

*Milk.*—Another great means of distribution of tubercle bacilli is the milk from tuberculous cows. The extent of this peril may be indicated when it is stated that at least 40 per cent. of milk cows in Great Britain are infected with tuberculosis, and that a great number of those supply milk containing tubercle bacilli. The germs may gain entrance to the milk by the udder of a cow being the site of a tuberculous lesion, or because the tuberculous process is disseminated throughout the body, and the bacilli, circulating in the blood, are excreted by the milk glands. Again, the bacilli may be discharged from tuberculous ulcers in the intestine and the infected fæces gain entrance to the milk. To those who are familiar with the conditions under which many of our cows are kept, and with the milking methods commonly in use, it will be at once appreciated how easy it is for infected excreta to gain entrance to the milking-pail.

*Tuberculous meat* may, to some extent, also spread the bacilli, but the danger here is greatly minimised because of the cooking which the meat undergoes before being partaken of. Tubercle bacilli in the centre of a large joint have been found to be alive after the ordinary cooking process,

but any superficial contamination of the meat is rendered innocuous by cooking.

Thus to a very great degree the prevention of the distribution of the tubercle bacillus is to be secured by the control of the consumptive patient and the tuberculous cow.

**Varieties.**—There have been recognised four distinct types of the tubercle bacillus:—(a) The Piscine or Ichthyc type; (b) the Avian type; (c) the Bovine type; (d) the Human type.

*Ichthyc Type.*—In certain diseases that are found in fish, lizards, frogs, turtles, etc., there are present “acid-fast” bacilli which are so closely allied to tubercle bacilli that they have been assumed to be a special variety of it. These bacilli grow rapidly at a temperature of about 20° centigrade, but do not grow at the temperature of the human body. They have never been found in man, so are only of interest in comparative pathology.

*Avian Type.*—This is found in certain diseases of fowls. It grows best at a temperature about 40° C. It is longer, and the colonies are moister on the surface, than the human type. Like the fish variety, it has never been found in man, so it is of no practical importance to the student of medicine.

*Bovine and Human Types.*—These are the only varieties with which we need concern ourselves. While there can be no doubt as to the nearness of their relationship, they can be shown to have quite distinct differences. The bovine bacillus is shorter, thicker, and less curved than the human bacillus. Its capsule is thinner, and it stains more readily. It grows slowly and feebly on glycerine agar, and forms a thin layer on the surface. For this reason it is spoken of as being dysgonic. The human type, in comparison with the bovine, grows more readily, forming a thick colony with rough puckered surface. Because of its relatively rapid growth, the term eugonic is applied. Experiments have been made on, among other animals, cows and rabbits with both of these types of bacilli. When the bovine type is injected into a cow in sufficient dosage it gives rise in about four weeks to a generalised tuberculosis, whereas the human type in a similar

dosage produces for the main part a local lesion. Again, in rabbits the bovine type causes general tuberculosis, while the human type forms local lesions slowly. If injected into the knee-joint of a cow, bovine bacilli within three or four weeks will produce an acute tuberculosis of the joint with erosion of the cartilage, and, later, will cause a generalised tuberculosis. The human bacilli, injected into the joint, produce a slight thickening of the synovial membrane, with an effusion of fluid into the joint, a condition closely allied to the tuberculous synovitis in the human being.

**Transmutability of the Bacilli.**—Much interest attaches to the question as to the possibility of the bovine and the human bacilli being one and the same: the variations in their effects on animals, and their differences in growth, being simply due to alterations in environment. Bacilli have been described by some observers, in which variations from either type have been noted, in cultural and inoculation experiments. Some observers have stated that they have succeeded in converting the bovine to the human type, and *vice versa*. Other workers, however, of equal repute have been unable to confirm these observations, and deny that any change had been noticed in the behaviour of either type of bacillus after passage, for several generations, through animals, or after prolonged subculture in artificial media. The question is not definitely settled yet. It is quite conceivable that, if the human type of bacillus were continuously passed through many generations of cows, ultimately the bacillus might show characters conforming closely to those of the bovine type. In the present state of our knowledge, however, we have to assume the fixity of type.

**The Relative Frequency of Infection of the Bovine and Human Types.**—Both bovine and human bacilli infect man. The bovine type is undoubtedly the source of a great amount of sickness in children, although producing very little among adults. In the year 1901 Koch startled the scientific world by announcing that the bovine type of bacillus was non-pathogenic to man, or at any rate so rarely communicated to man that preventive measures against it were unnecessary. This statement attracted much attention as, if it proved to be correct, there was no necessity to be concerned about

tuberculous cows or tuberculous milk. So much importance was attached to the matter in Great Britain that a Royal Commission was formed to substantiate or disprove his statement. Before the Commission had progressed far with its investigation it was satisfied that bovine tubercle was responsible for a considerable amount of disease, especially amongst children. Since that time a large amount of work has been accomplished along this line of investigation, and the findings of the Commission established beyond all doubt.

The position stands somewhat as follows at present. In *meningitis* the majority of the cases are due to the human type, the bovine type being responsible, according to some workers, for about 18 per cent.; but Novick, writing in the *Journal of Medical Research*, states that in 48 cases of meningitis he found 3 due to the bovine bacillus. In *generalised tuberculosis* the percentage due to the bovine bacillus is 16 per cent. In *primary abdominal tuberculosis* the percentage due to the bovine type increases remarkably. For all ages that type causes about 50 per cent. of the cases, and in children about 60 per cent. This is as we should expect, considering the chances of infection by tuberculous milk. In *bronchial glands* the results of 18 cases collected by Cobbett were 17 human and 1 bovine.

In the *cervical glands* which drain the naso-pharynx and tonsillar regions we find the proportion of cases due to the bovine bacillus rises again. There is a considerable variation in the incidence for different countries, but the average works out about 50 per cent. bovine in the cases under sixteen years of age, and at about 75 per cent. in cases under five years. As evidence of the wide variation which exists, some German observers recorded a total percentage of 33 in children under five years, whereas Mitchell, working with Edinburgh children of the same age, found no less than 90 per cent.

In *bone and joint* cases a somewhat similar discrepancy exists. Fraser gives his results in Edinburgh children as approximately 60 per cent., but Griffith, working on Scottish children, only found 29 per cent. due to the bovine bacillus.

Griffith's figures are worthy of being set out in full detail:—

Ages.	No. of Cases.	Human.	Bovine.	Atypical.
0-5 . . .	47	31	14	2
5-10 . . .	108	75	31	2
10-15 . . .	62	52	7	3
15-25 . . .	15	12	3	...
Over 25 . . .	29	26	...	3
	261	196	55	10

All ages . . . 55 out of 261 = 21 per cent. bovine.

Under 10 years . . . 45 „ 155 = 29 „ „

Over 10 years . . . 10 „ 106 = 9.4 „ „

In another series of cases, in which the material for examination was produced from England and Scotland, the result is as follows:—

	Cases.	Human.	Bovine.	Atypical.	Bovine.
					Per cent.
England . . .	109	88	16	5	14.7
Scotland . . .	27	19	8	...	29.6

It will clearly be seen that Koch's statement has been conclusively proved to be wrong in respect of all non-pulmonary forms of the disease. It is right, therefore, that precautionary measures should be taken to prevent children from being infected by the bovine bacillus.

However, when we come to discuss the relative frequency of the two types of bacilli in lung lesions we might well-nigh agree with Koch. In tuberculosis of the lungs the human type is found in about 99 per cent. of the cases, so that for all practical purposes when we think of pulmonary tuberculosis we think of the human type of bacillus. The eradication of tuberculosis in childhood, a time when the glands and bones are chiefly infected, calls for a supply of tubercle-free milk, and the protection of the child from close contact with the individual suffering from pulmonary tuberculosis.

At the same time it should be noted that infection of the



child by bovine tubercle does not give rise to pulmonary tuberculosis in adult life, nor, as far as we know, does a bovine infection confer any immunity against pulmonary tuberculosis. Certain writers have stated, though as far as I can judge without the slightest foundation whatever, that in Great Britain we owe our comparative immunity to pulmonary tuberculosis to the fact that the majority of us have already been inoculated in youth by the bovine type. From such statements it would appear to be desirable that in order to prevent consumption in the adult, all children should be carefully supplied with milk containing bovine bacilli. This counsel will not, it is believed, recommend itself to the majority of practitioners. The damage done by the bovine bacillus is real, its protective value problematical.

**Other Acid-fast Bacilli.**—For some time after the discovery of tubercle bacilli, it was thought that the power of resisting decolorisation by acids was peculiar to the tubercle bacillus, but later this was found to be incorrect. Many other bacilli have this power. Indeed there is a fairly large group of bacilli known as the acid-fast group. The terms tuberculoid, or pseudo-tubercle, have also been applied to this group, and it is computed that there are at least forty different micro-organisms which have the acid-resisting power. The best known of these are the smegma bacillus, the Timothy grass bacillus, the butter bacillus, Johne's bacillus, and the lepræ bacillus. The fact that these "acid-fast" organisms exist has not, up to the present, been found to impair the clinical significance of the finding of an acid-fast bacillus in any excretion from the human body. Given an acid-fast bacillus in the sputum, it is invariably assumed that the patient is suffering from tuberculosis. Mistakes have been made in the case of the smegma bacillus, and it is recorded that kidneys have been excised needlessly because of the presence of these bacilli in the urine. The tubercle bacillus is alcohol- and acid-fast, and is distinguished in this way from the smegma bacillus, which is decolorised by alcohol. When injected into animals these pseudo-tubercle organisms cause tissue changes identical with those resulting from the inoculation of tubercle bacilli, but unlike the true tubercle the lesions remain localised and do not tend to break down.

## CHAPTER IV

### SPREAD OF INFECTION

THE source of tuberculosis being an infected man or animal, it is now necessary to consider how the bacillus is conveyed from the infected to the healthy. Infection by the bovine bacillus is practically always the result of infected milk. In infection by the human type of bacillus the germs are carried by the air, either by infected dust or by minute droplets of sputum or saliva.

(1) **Dust Infection.**—It was formerly assumed that the only way in which the disease was spread was by infected dust. This was caused by the careless consumptive spitting on the floor, and the sputum, after undergoing desiccation, being ground into powder by the traffic in the room. Afterwards, when the floor was swept, the infected dust was stirred up, disseminated through the air of the room, and inhaled by any individual in the room. That tuberculosis can be caused in this way has been demonstrated by keeping animals in a chamber where there was infected dust, which was occasionally stirred so as to raise it into the air of the compartment. Guinea-pigs, under such conditions, always became tuberculous. While doubts have been expressed as to the probability of this method of infection on the ground that it is an extremely difficult matter so to dry sputum that it can be powdered into dust, it must be admitted that sputum on the floor of a room constitutes a real menace to the healthy inhabitants of that room, and that infected dust plays a part in the spread of the disease.

(2) **Droplet Infection.**—The other method by which the bacilli may be conveyed from the sick to the healthy is by expulsion from the mouth of minute droplets of saliva or

phlegm which carry with them tubercle bacilli. It is a well-known fact that in forcible speaking or coughing a fine spray of mucus is expelled from the mouth, and that this spray floats in the air of the room to a considerable distance before it is deposited. One observer, after rinsing his mouth with an emulsion of *Bacillus prodigiosus*, spoke forcibly for some time in a room in which he had exposed some sterile culture plates, and then, on carefully covering the medium which he had exposed and incubating it, he found that the *Bacillus prodigiosus* developed on all the exposed plates. The writer has asked a patient to cough while holding a clean glass slide in front of his mouth, and on staining that slide demonstrated the presence of tubercle bacilli. While much has been written on the relative frequency of dust or droplet infection, both undoubtedly play a part in the production of tuberculosis. It is certain that a workman, working at the same bench with a consumptive who is frequently coughing, runs a considerable risk from his infective neighbour. Indeed, there are numerous cases on record where the individuals date their illness from the time they worked beside a tuberculous patient, but whether the infection was by dust or droplet, or both, is difficult to prove.

**Portals of Infection.**—We have now to discuss what are the precise channels by which the bacilli gain entrance to the body. The organisms may be in milk or in the air, and so infection may take place by the digestive or respiratory tracts. They also find entrance by other channels, and the relative frequency of infection by these various pathways will now be considered.

(1) *Pre-natal Infection.*—Pre-natal infection is a very rare occurrence, but it takes place. Bang, after examining a large number of calves, states that congenital tuberculosis in these animals occurs in about 1·4 per cent. In the human subject it is probably rarer. The infection may be in the *ovum* itself, but such an occurrence must be very occasional. Tuberculosis has been produced experimentally in birds by inoculating the egg with tubercle bacilli and demonstrating bacilli afterwards in the body of the chick, thus showing the possibility of infection by the *ovum*. Theoretically it is possible that the bacilli might be conveyed to the *ovum*

by means of infected *sperm cells*, but probably such an occurrence never takes place.

Pre-natal infection is more likely to be of *placental* origin. Several cases are on record where the placenta of a tuberculous mother was shown to be infected. It is probable that if diligent search were made, tuberculosis of the placenta would be found to be more common than is supposed. In the present state of our knowledge we have proof that congenital tuberculosis occurs, but is apparently so rare as not to be of much practical importance.

(2) *Post-natal Infection*.—Infection after birth may take place through the skin, the respiratory or the alimentary tract, or tonsillar region.

(a) *Infection by Skin*.—This is almost always a wound infection, as the normal skin is an efficient protection against invasion. It is most commonly seen on the hands of butchers or knackers, and occasionally it is found on the hands of veterinary surgeons employed at the large municipal slaughter-houses on meat inspection. There are a few cases on record where nurses have infected their hands by cutting them accidentally with broken sputum mugs. The disease in these cases is usually local and takes the form of the so-called post-mortem wart. However, in some cases the primary sore has been followed by lymphatic infection and later by pulmonary disease.

(b) *Invasion by Respiratory or Intestinal Tracts*.—As the lungs are the organs most frequently attacked by tuberculosis, it was formerly assumed that the bacillus gained entrance by inhalation and that this was the only portal of infection. This opinion held undisputed sway for a long time. About the year 1903 von Behring disputed its accuracy, and stated that tuberculosis of the lungs was due to the ingestion of the bacillus. Two years later Calmette declared that by far the greatest number of cases of pulmonary tuberculosis was of intestinal origin. An immense amount of work has been done in the attempt to settle this question. Calmette performed several interesting experiments which appeared to support his claim. He fed guinea-pigs with a mixture containing china ink, and demonstrated later, as he thought, the pigment in the lungs of the animals. At the same time,

however, he admitted that he could not find any pigment in the lungs of young guinea-pigs so fed. The true significance of this exception is seen later in Cobbett's experiments. Some other observers agreed with Calmette and held tenaciously that pulmonary tuberculosis was of intestinal origin. Other observers, however, were unable to confirm Calmette's observations, but we need only note Cobbett's work.

This investigator was unable to produce any pigmentation of the lungs although he used much larger quantities of china ink than Calmette did. He had taken the precaution to use control animals, and he noticed that in the older animals used, the controls would sometimes show some pulmonary pigmentation. This pigmentation, he discovered, was not present when country-bred animals were used, so he concluded that in the town-bred guinea-pigs the pigment was the result of the soot in the air, just as the lungs of city dwellers show more pigment than do the lungs of those living in the country. This explains the difficulty that Calmette met with when he found that he could produce no pigmentation of the lungs of young guinea-pigs. The city of Lille, in which Calmette worked, has a smoky atmosphere, so that the lungs of older animals reared in that town would be pigmented like those of town-dwellers, that is, they would have some soot deposited in them. Calmette was of opinion that the air was efficiently filtered by the nostrils, but this is not so, for Cobbett sprayed an emulsion of the *Bacillus prodigiosus* in front of a guinea-pig, which was then killed, and an examination showed that the bacilli had within five minutes been conveyed to the air vesicles at the remote edge of the lung. The result of all the recent reliable experiments is to establish, more firmly than ever, the fact that the respiratory tract is the main portal of infection in lung tuberculosis.

It has, however, been demonstrated that after feeding an animal with an emulsion of tubercle bacilli the bacilli may be found in the thoracic duct, so that while the respiratory tract is the usual portal, the possibility of the intestinal mode of entrance is not altogether excluded. The greater number of bacilli gaining entrance into the lymph-stream of the



abdomen are probably caught in the mesenteric glands; only a few succeed in passing.

(c) *Other Channels of Infection.*—The frequency of tuberculosis of the cervical glands would seem to prove conclusively that the bacillus often gains entrance through the tonsil or pharyngeal mucous membrane and infects the nearest lymph glands. The opinion has been expressed that decayed teeth or the conjunctival sac may form other portals of entry. Bacilli which gain entrance through the tonsils may remain localised in the glands and cause little or no trouble, or they may infect gland after gland until in the end they gain entrance to the blood-stream and are then carried to the lungs. At other times gland after gland is involved until one lying adjacent to the apical pleura is infected, and this, giving rise to a pleurisy with resulting adhesions between the two layers, may enable the bacilli by direct spread to infect the lung itself. Some observers have stated that there is a direct connection between the deep cervical glands and those of the root of the lung, and so the infection of the cervical glands may thus pass by direct continuity to the bronchial and root glands. Thus by one portal or another do these bacilli gain entrance, and in the following chapter we shall consider their behaviour and results when in process of growth in the body.



## CHAPTER V

### PATHOLOGY

WHEN the tubercle bacillus is implanted successfully in the tissues it produces a characteristic result. There appears to the naked eye a small nodule, or tubercle as it is called, about the size of a millet seed, apparently round in shape, semi-translucent or greyish in colour. On more minute examination this is shown to be composed of a group of minute tubercles, a single one being invisible to the naked eye. In the lung the first deposit is commonly in the wall of a bronchiole. As the tubercle becomes older it shows a yellowish opacity in the centre. Microscopically the tubercle consists of a rounded mass of epithelioid cells with, as a rule, one large giant cell in the centre. The epithelioid cells have oval nuclei and faintly staining bodies. Supporting them is a delicate network of fibrous tissue and fibrin. The giant cell contains many nuclei which are arranged towards its periphery. The origin of these cells is not quite settled. It has been variously contended that the epithelioid cells are the result of proliferation of the fixed tissue cells, or that they are due to the invasion of white blood corpuscles. The origin of the giant cell is also uncertain. It has been thought to be due to the division of a cell nucleus without the protoplasm dividing, or to the absorption by a single cell of the nuclei of surrounding cells the bodies of which have disappeared.

Around the edge of the tubercle there is a well-marked round-celled infiltration. Tubercle bacilli are usually found in the giant cell and among the epithelioid cells. The whole structure of the tubercle is avascular and no new vessels are formed in it. Partly as a result of this, and partly also owing to the toxins of the bacillus, the viability of the tubercle is not great and it may soon begin to degenerate. When it

does so, it becomes yellowish in colour and caseous in consistence, and the cells gradually lose their characteristic appearance until in the end no definite structure can be seen at all when examined microscopically. A further degenerative process which the tubercle may now undergo is the liquefaction of the central caseous material. This is thought to be due to the action of some unknown cell ferment. Subsequent to liquefaction the tubercle may burst into the neighbouring bronchus or pulmonary vesicles, and this leads to the further spread of the disease.

**Mode of Spread.**—The disease may be propagated in the lung by four methods : (1) by discharging into a bronchus (aerogenous); (2) by contiguity, that is, gradually spreading into the neighbouring tissues; (3) by the lymph-stream (lymphogenous); (4) by the blood circulation (hæmatogenous).

(1) *Aerogenous Spread.*—The primary tubercle, caseating and liquefying and then discharging into the bronchiole, scatters an infective material which, by the process of respiration, may be aspirated into the neighbouring vesicles, or expelled along the bronchiole with the natural secretion. Two things may happen. The infective material may set up in the vesicles a small patch of caseous pneumonia; or, in the walls of the vesicles or bronchioles, another fresh crop of tubercles may be produced to undergo, in their turn, a degenerative process similar to that of the primary tubercle. By these means there is formed an airless mass in the lung, often with a small central excavation, and with the surrounding alveoli filled with inflammatory, caseating material, and tubercles in the vesicle walls.

(2) *Spread by Contiguity.*—The second mode of spread is by contiguity, that is by fresh tubercles forming at the edge of the old caseating nodule. By successive crops growing, coalescing, and degenerating, a massive consolidation with central excavation is ultimately produced.

(3) *Lymphogenous Spread.*—This is the result of the lymph channels being invaded by the organism, which is then carried along by the lymph-stream. As a rule the bacilli are arrested at the first lymph gland if they have not already attacked the lymphatic wall itself. In the root gland they may settle and grow, causing enlargement and caseation of the gland, or they

may be destroyed. It is an accepted fact that bacilli may pass through the mucous membrane of the lung and leave it intact or produce a lesion so small as to escape detection, and yet these bacilli may gain entrance to the lymph channels and pass on, to be caught in the glands and produce tuberculosis of these structures.

(4) *Hæmatogenous Spread*.—Again the bacilli may be distributed by the blood-stream. A tuberculous ulceration may involve the wall of a blood-vessel and the caseating process erode the vessel wall so that the infective contents are discharged into the blood-stream. If the vessel be a vein, the distribution is widely spread, and the consequence may be disastrous, as the whole systemic circulation is involved, and the result may be acute miliary tuberculosis.

**Reparative Processes.**—While the natural tendency of the newly formed tuberculous nodule is to caseate and liquefy, other processes are almost invariably at work. There is an attempt, and often a successful one, at the formation of fibrous tissue which may limit the spread of the disease and ultimately encapsule the focus altogether in scar tissue. In fact, in every case of chronic tuberculosis there would appear to be a race between the degeneration and liquefaction of the tuberculous nodule on the one hand and the production of encapsulating fibrous tissue on the other. Whichever process wins the day, decides the fate of the patient. When the focus is small, and the reparative process active, the disease may be healed completely without any suspicion being raised that the person has been subject to tuberculosis. On the other hand the caseous nodule may be incompletely embedded in fibrous tissue, and may remain in this condition for a long period, a continual source of danger to the patient, as for an indefinite time it contains live tubercle bacilli. These may be stirred into activity in many ways, all of which we do not thoroughly understand, but among which may be mentioned repeated catarrhal attacks of the lungs, acute infections, or other debilitating causes. In other cases in which the caseous nodule is thoroughly encapsuled, its contents slowly become desiccated and impregnated with lime salts so that there is left a calcareous mass embedded in fibrous tissue.

Reparative processes may take place, and take place successfully, at any stage of the disease. Even when destruction of lung tissue has occurred, the disease may be completely arrested and the patient left with a smooth-walled cavity lined on the inside with dense fibrous tissue. As a rule, when there is extensive development of fibrous tissue the chest wall is retracted considerably, with hollowing, more or less pronounced, above and below the clavicle, and in the more extensive cases with drooping of the shoulder. In all cases of chronic disease there is an increase of the connective tissue stroma of the lung, and in some this increase becomes so marked and extensive that the caseous degeneration forms quite a secondary element, and we have the type of disease known as fibroid phthisis.

**Site of Lesion.**—In chronic pulmonary tuberculosis of the adult the primary seat of the disease is in the apex, and more often in the right than the left. Fowler's observations on this point are noteworthy. He found that the primary focus is not in the extreme apex but from one to one and a half inches below this, and nearer the posterior surface than the anterior. For this reason the physical signs are early observed in the supra-spinous fossa. This area corresponds with the supra-clavicular fossa in front. From the primary focus secondary deposits are formed in the anterior part of the lung, and these tend, by a process of infiltration, to spread downwards and outwards, and the physical signs appear in the outer part of the first and second interspaces. Before long, evidence of tuberculous deposit will be found in the apex of the lower lobe. The deposit is again about an inch or so from the actual apex, and in situation corresponds roughly to a point opposite the 4th or 5th dorsal vertebræ midway between the spine and the edge of the scapula. From this the disease tends to spread downwards and outwards along the margin of the interlobular fissure, and slowly infiltrates the whole of the lower lobe. Before the process has extended far into one lung the apex of the other becomes involved. For some not very well-known reason the middle lobe on the right side is only affected late in the disease and sometimes not affected at all.

**Gross Pathological Appearances.**—In a case of advanced tuberculosis the emaciation of the cadaver is noteworthy.

There is, in extreme cases, an almost complete absence of subcutaneous adipose tissue. The affected lungs are often firmly, and extensively, adherent to the chest wall: so firmly that they require to be cut away, and in some cases so extensive that the pleural sac is totally obliterated. The pleura being often enormously thickened, the lung is much contracted and the diaphragm pulled upwards on the affected side. On cross section there may be found, in the apex, one or more cavities of varying shape. Some may be smooth-walled and apparently healed. Others may be partially filled with caseous material or broken-down debris with ragged disintegrating walls. Running across the cavities or hanging loosely into them are strands of fibrous tissue. Some of these strands may be the remains of blood-vessels which have so far resisted the destructive process. Some indeed of these vessels may be patent, others quite occluded, owing to an obliterative endarteritis. Other strands in the cavity may be the remains of the thickened fibrous structure of the lungs.

Below the area of cavitation there is often seen a broad band of material, partly grey and partly yellow in colour, forming a zone of caseous pneumonia. Below this there may be fairly normal lung with a caseous nodule here and there. The glands at the root are always swollen and enlarged, and some of them may be caseous.

In chronic cases, with a marked formation of fibrous tissue, there may be sacculated or cylindrical dilatation of the bronchi. In these cases also the remaining portions of both lungs show a considerable degree of emphysema. In some instances where one lung is much contracted, and the other fairly healthy, the emphysematous margin of the latter will be found extending well over the middle line into the other side for a varying distance of one or two inches. This can often be made out during life by careful physical examination. In cavity formation there will frequently be found aneurismal conditions in the vessels which run across the cavity or in those which are partially embedded in the thick fibrous tissue in the cavity walls. The aneurisms may be small or miliary in character, or there may be one or more of moderately large size extending up to two or more inches in diameter.





FIG. 8.—Lung showing extensive cavitation and consolidation.





## CHAPTER VI

### PREDISPOSITION TO TUBERCULOSIS

IT is a well-established fact, as was shown in the chapter on Epidemiology, that over 90 per cent. of the population is infected with tuberculosis by the time they reach the age of 14, and yet on examination of the death-rates it is seen that the cause of death is certified as due to tuberculosis in only about 10 per cent. This demonstrates that in the community generally there is a certain widespread and active resistance to the development of the tubercle germ, or in other words that the 10 per cent. who die from tuberculosis have been predisposed to the disease from one cause or another.

There are two aspects from which this might be considered: (1) that of the bacillus itself; (2) that of the person in whom the germs are planted. Or, to put it in another way, it can be discussed from the point of view of the seed and the soil.

(1) **The Bacillus.**—It is a common observation in experimental bacteriology that if a minute dose of tubercle bacillus is injected into a susceptible animal the resulting lesion is localised. If a massive dose is given, however, the disease becomes generalised and the initial local lesion is only a part of a general tuberculosis. While it may be unsafe to treat the results of experiments of this character on animals as always applicable to man, still it may be accepted that different results arise from the administration of different quantities of the bacillus. It is to be expected that when massive doses of tubercle bacilli gain entrance to an individual he is more likely to fall a prey to tuberculosis than if he received only minute doses. The practical bearing of this is at once seen. An infant who is nursed or fondled by a

## PREDISPOSITION TO TUBERCULOSIS

consumptive mother is exposed to massive doses of infection, and is almost certain to succumb to the disease, whereas in the case of a child exposed to an occasional small dose the result may only be a local glandular involvement. This point of view, namely the danger of mass infection, should always be remembered when dealing with a consumptive in an actively infectious condition in a small house where the facilities for isolation do not exist. The risks of the other persons who live in the house becoming infected are numerous and serious.

In addition to the dose of the bacillus the degree of virulence of the organism inoculated has to be considered. Having regard to the very protean character of tuberculosis, there can be little doubt that different strains of bacilli exist, some of which are more virulent than others. This may be somewhat difficult to prove experimentally, but the clinical evidence which we see daily may help to bear this point out. One case of tuberculosis of the lung may be seen in an early stage and pass on to a complete recovery, while another seen in a similar stage may, in spite of all remedial measures, proceed to a fatal issue. Unfortunately for the certainty of the above theory, the virulence of the bacillus is not the only point to be considered; that other unknown quantity, viz. the resistance of the patient to disease, comes into operation, and introduces a doubt as to whether the virulence of the organism accounts for all the clinical varieties of tuberculosis. In the case of some patients who have had tuberculous cervical adenitis, if their lungs become infected at a later date, they show a pulmonary tuberculosis of a very slowly progressive nature. Some observers have been of opinion that in these cases the bacillus has become of a less virulent character, but it might just as strongly be upheld that the slow progress of the pulmonary disease is due to the fact that the individual has already acquired a high degree of resistance to the organism. This whole question demands further research.

As neither the dose of infection nor the virulence of the bacillus can explain all the clinical varieties of tuberculosis, the second point has to be considered, viz. :—

(2) **The Individual.**—*Heredity* plays an important part

in the development of pulmonary tuberculosis. It is an accepted commonplace fact that certain families are more prone to be affected by tuberculosis than others, and it is generally believed that those families have in some way or other a lessened resistance to the disease which they have inherited from one or other parent. The statement has even been made that the type of tuberculosis is apt to be hereditary—that is, if a parent be affected with tuberculosis of bone, then should any of the offspring become infected, the chances are that the tuberculosis will be in bone. Again, if the parent suffer from a right apical lesion, should any of the offspring become affected with pulmonary tuberculosis, the chances are that the right apex will be again involved.

What the factors are which produce this hereditary predisposition to tuberculosis we do not know. Much work has been done on this point, but so far it has produced little certainty of knowledge. It has been suggested that there is a lowering of the protective properties of the blood serum because of a deficiency of agglutinins, precipitins, etc., but this has not yet been fully proved. Other observers, particularly those of the French school, have stated that there is a lack of lime salts in the blood content, and that this deficiency is due to the fact that there is an abnormal calcium excretion in the urine of the individual predisposed to tubercle. The reduction of the lime salt is supposed in some way to favour the advance of the tuberculous process. This theory has not been accepted by all workers in this field. It has also been thought that the hereditary predisposition might be due to defective secretion in some of the ductless glands, such as that producing hypothyroidism. The whole matter in regard to hereditary predisposition to tuberculosis has been summed up by stating that there is apparently a lack of some of the biochemical defences of the organism, the nature of which is not yet understood.

**Acquired Predisposition.**—As in the case of the classical sower who went forth to sow, when some of the seed fell on good ground, so the tubercle bacillus at times falls into good ground which has been prepared by one means or

another. This predisposition to the disease is acquired in various ways, and these will now be considered.

(1) *Environment*.—The individual's surroundings play an important part in his defence against tuberculosis as well as against other diseases. City dwellers, for instance, are more prone to tuberculous disease than are dwellers in the country. This is well shown by the figures supplied by the Registrar-General for Scotland, in which year after year he shows the various rates in large towns, small towns, and country districts: and each year the large towns have the heaviest incidence of disease. In the towns themselves the influence of the environment is further seen. Taking for the purpose of comparison two wards in the city of Edinburgh, one containing a considerable portion of crowded, somewhat slum, property, and the other in a good residential district, it is seen that in the former area in a given year the notification rate per 1000 was 3.3, the death-rate 1.3, whereas in the better class ward in the same year the notification rate was 1.2 per 1000, the death-rate 0.5. Thus, where the environment is bad the death-rate is nearly three times as great as in the district where the environment is good.

(2) *Social Conditions*.—The actual environment, however, is not the sole factor which is concerned in the predisposition to the disease. The social condition of the individual affects it to as great an extent as environment does. Poverty is one of the great factors in the causation of this disease, and its influence on the course of the disease is such that it has been well said that no poor man can afford to have pulmonary tuberculosis. Poverty carries in its train all those conditions which make for a low standard of vitality, with a consequent lessened resistance to disease. It compels an individual to live in a poor part of the town where houses are small and crowded together, and often causes gross overcrowding in the house itself. Poverty keeps the person just on the margin of a bare subsistence line, and oftentimes he is even below that. It is unlikely that a disease-resisting individual can be reared under such circumstances. In addition, the poverty-stricken part of the community is frequently on a lower educational level than the other members of the community. They are oft-times ignorant of the laws of health, even of the

most elementary laws of ventilation and cleanliness. The ignorant are careless and indifferent as to the dangers from infected expectoration, and their habits are such that opportunities for infection exist abundantly. It appears certain that the battle against tuberculosis will have a greater measure of success when we are dealing with a better-educated and a better-cared-for proletariat.

(3) *Habits*.—The personal habits have also a marked influence on predisposition. Excessive indulgence in alcoholic liquors has a most profound bearing, both as regards the individual himself and those dependent on him. A drinker, generally speaking, has a poor resistance to all diseases, and when he is attacked by tuberculosis the chances of a successful resistance to this disease are small. In its relation to children who are dependent on the drinker, it has to be borne in mind that the money expended on alcohol is, as a rule, money that should have been spent in the care and nurture of those children. Thus they suffer from lack of proper sustenance, and this, bringing about a low standard of vitality, makes them more prone to tuberculosis.

(4) *Occupation*.—It is well known that some occupations predispose more to tuberculosis than do others. As a general rule it may be taken that an indoor occupation gives rise to more tuberculosis than does an outdoor one. Again, dusty indoor occupations predispose more to tuberculosis than do those where dust is not a prominent feature. Yet again, in those occupations where the dust is of a hard, gritty, irritating character the incidence of tuberculosis reaches its highest mark.

All dusty occupations are not equally prone to produce tuberculosis; for instance, a coal miner is not so liable to tuberculosis as the Cornish tin miner, nor is the worker in limestone so much affected as the worker in hard sandstone. Collis, in an exhaustive study of this question, has shown that it is the dust containing crystalline silica which is the worst, and he has demonstrated that in any industry where such dust is generated, the outdoor or indoor occupation is of secondary importance, and that it is the silica dust itself that is the prime factor. Tatham showed very convincingly the influence of occupation upon tuberculosis by giving the



comparative mortality figure of a few occupations, taking agriculturalists as the standard.

Agriculturalists . . . .	100	File makers . . . .	373
Cotton operatives . . . .	244	Cutlers . . . .	407
Lead workers . . . .	247	Potters . . . .	453
Glass makers . . . .	335		

Again Collis confirms this by the following table :—

*Comparative Mortality Table in Certain Occupations, 1900-2.*

Class.	Phthisis.	Class.	Phthisis.
All males . . . .	186	Innkeeper, publican . .	232
Labourer in agricultural districts . . . .	75	Tailor . . . .	243
Coal miner, Lancashire . .	98	Shoemaker . . . .	247
Wool, worsted operative . .	161	Potter . . . .	291
Hosiery operative . . . .	200	Printer . . . .	323
Cotton operative . . . .	214	Cutler . . . .	506
		Tin miner . . . .	851

**Other Diseases predisposing to Tuberculosis.**—It is probable that no disease is more blamed as the cause of tuberculosis than *influenza*. With recurring monotony the statement is made by patient after patient, "I was all right until I had influenza some months ago." The reason for this is that influenza is a very convenient term for all febrile conditions of unknown origin. If a patient is suffering from some feverish attack what more convenient name for the illness than influenza? If a good recovery is made, well and good. If convalescence is retarded, what else could be expected from a bad attack of such a disease? There is no doubt that very many of the so-called attacks of influenza are simply acute exacerbations of already existing pulmonary tuberculosis. In all supposed cases of influenza, at a time when there is no epidemic, a careful examination of the lungs should be made. This will often reveal the true, tuberculous nature of the illness. When this examination is done as a matter of routine, we shall hear much less of influenza as a cause of tuberculosis. At the same time, there is good ground for believing that many cases of tuberculosis have their commencement in an attack of influenza. It is quite likely that the tubercle bacilli may have been already implanted, but that the natural resistance

of the individual prevented their growth and spread, and when this resistance is lowered by the toxin of influenza, then the tubercle bacilli are enabled to grow and multiply. It should be stated, however, that there is a considerable divergence of opinion on the part played by influenza in the causation of tuberculosis.

*Whooping-cough and Measles.*—These two diseases are the undoubted starting-point for many cases of tuberculosis in children, especially when they are complicated by bronchitic conditions during the acute illness. After measles especially, if tuberculosis ensues, it is not uncommon that the disease is of the acute pneumonic type. In measles there is a lowering of the resistance to tuberculosis which can be shown by the absence of the skin reaction to tuberculin. A child who will ordinarily give the Von Pirquet reaction, thus showing that it has been already infected by the tubercle bacillus, will not show it during or for some time after an attack of measles. If we take the reaction as meaning that the tuberculin combines with certain antibodies which are in the cells or blood-stream, and that this new combination gives rise at the site of inoculation to an area of inflammation, then after measles, when this reaction does not appear, we are driven to conclude that the antibodies are either not present in the child's system or are in an inactive state. The cells have either been unable to form the antibodies or the toxin of the measles germ has for the time being neutralised their effect. Be that as it may, the fact remains that measles is especially apt to be followed by tuberculosis. The practical point in dealing with measles is, of course, to see that a thoroughly good convalescence is established; and were the after-care of measles more thoroughly dealt with, we should hear less of tuberculosis following this disease. With regard to whooping-cough, the paroxysmal strain on the respiratory organs, coupled with the debilitated state to which it always reduces the patient, tend to activate any latent focus of tuberculosis present in the child's lungs.

*Recurring Bronchial Catarrhs.*—Many patients who suffer from tuberculosis have complained that they have been the subject of frequent nasal, laryngeal, and bronchial

catarrhs before the onset of the disease. These may be looked upon as indications that the individual possesses a mucous membrane which is more than usually susceptible to the attacks of the ordinary catarrhal organisms, and it is probable that the resistance to the tubercle bacillus is also lessened.

*Bronchitis and Emphysema.*—These two conditions are often associated with tuberculosis, and this is especially so in the old patient of the Poor Law class. The writer is not of opinion that the bronchitis or the emphysema predisposes to tuberculosis. In many cases the diseases run concurrently, but in others the emphysematous condition is a result of the coughing produced by the tuberculous process. It is extremely difficult in a well-marked emphysematous chest to exclude the presence of tuberculosis by physical signs. Time and again I have presented to my students for examination patients with well-marked emphysema in whose sputa there were abundant tubercle bacilli, and invariably the diagnosis has been made as bronchitis and emphysema. This was not to be wondered at, as there were few localising signs of tuberculous disease and these were hidden by the emphysematous changes. The emphysema, however, often influences the tuberculous process in a profound degree. The course of the disease becomes more chronic and there is less tendency to breaking down on the part of the diseased tissue. Fibrosis predominates.

**Local Predisposing Causes.**—The favourite site for the development of tuberculous lesions in adults is the apex, and much work has been done to try to determine why there is this special predisposition. Various theories have been advanced. *Stenosis of the upper thoracic aperture*, due to an ossification of the first costal cartilage with a consequent shortening of the first rib, was advanced by Freund many years ago. He showed that in persons who had died from tuberculosis there was a narrowing of the thoracic aperture on the affected side. Freund also assumed that the lung was affected because of the pressure of the first rib on it, and that this pressure interfered with the circulation, and with the proper emptying of the secretion out of the

underlying bronchioles. As a result, he argued, tuberculosis was more prone to develop at this spot. In an attempt to confirm Freund's theory the apex of an animal's lung has been constricted artificially by the introduction of a metal ring, and it was found that where the apex was constricted in this artificial manner and the animal inoculated with tubercle bacilli the disease developed in the portion of lung underlying the metal ring. In this way the observer was enabled to produce apical tuberculosis in either apex at will. It should be pointed out that such an operation has little or no bearing on the natural pressure exerted by the first rib. Freund's theory, it should be also noted, is not widely accepted as proven. Some anatomists are of opinion that the stenosis of the thoracic aperture is secondary to, and not the cause of, apical tuberculosis.

The usually accepted opinions as to why the apex should be attacked are: (1) that in the extreme apices there is a *lessened respiratory effort*, at any rate in adults; (2) that there is a *diminished blood supply*; and (3) that there is a *comparative lymph stasis*. All these three factors are contributory to the implantation and growth of any tubercle bacilli which may be brought to that area, either by the blood-stream or by the inspired air. As bearing on this theory, it should be noted that in children, in whom the respiratory movements are much more active and the blood circulation more rapid, the apex is not specially selected as the site of tubercle. It has been suggested that there may be, in the apex itself, some deficiency of tissue resistance to the organism such as does not obtain in the other parts of the lung. This is, of course, mere conjecture, and has not the slightest basis on which to rest. In the meantime we have to content ourselves with the theory of lessened blood, lymph, and air movement at the seat of election.

## CHAPTER VII

### HISTORY OF ILLNESS

THE successful implantation and growth of the bacillus brings about a chain of events which ultimately cause the patient to seek, or to be brought for, medical advice. It is the physician's business to determine, if possible, whether they are due to the active growth of the tubercle bacillus or to other causes. All due care should be exercised, as a diagnosis of "consumption" must not be lightly made.

**Family History.**—In investigating a case of illness of a possible tuberculous character, inquiry should always be made into the family history. Should it be found that there is a family tendency to tuberculosis, due weight must be given to that fact in forming an opinion at the end of the examination. Slight symptoms in a member of such a family acquire an importance which they do not possess in a patient with a good family history. As illustrating the extreme susceptibility of some families to tuberculosis, a family of seven has been known to be exterminated by the disease. The children reached early adult life, and one after another they were attacked by pulmonary tuberculosis and all died of it. The youngest and last survivor of the family was treated in a well-conducted sanatorium, but in spite of every care and attention the disease proved fatal.

In one consecutive series of 837 cases under my own care, a tuberculous family history was found in no less than 344, or 41 per cent.

Looking to these facts, a serious view should be taken of trivial complaints of ill-health in any member of such a family. Seemingly unimportant symptoms may be the first danger signals of the malady.



**Exposure to Infection.**—When examining the family history, should a case of tuberculosis be discovered in it, an attempt should be made to find out if the patient had been exposed to risk of infection from that case, and, if so, to what extent. In many instances a patient may have a bad family history and yet may never have been exposed to the risk of infection from sufferers. But it may happen that the risk of infection has been great, as in the case of a daughter nursing a tuberculous mother, sharing the same room or even the same bed for many months. In such a case the prolonged exposure to infection would be of much importance in forming a judgment as to the presence or absence of tuberculosis in any suspicious illness of the daughter. In the case of children the diagnosis may rest, in great part, on the question of exposure.

**Occupation.**—The question of occupation has an important bearing in helping to form a diagnosis. This has already been dealt with, to some extent, under the predisposing causes. The physician must not rest content with knowing the present occupation, but must examine carefully into previous occupations. A patient at the time of examination might quite well be a gardener and yet previously have worked for a considerable time in the gold mines of South Africa. This latter fact would influence the diagnosis considerably. Puzzling anomalous signs in the chest of an open-air worker, such as a carter, may suddenly become perfectly plain when it is discovered that the carter had been previously for many years a stone-mason.

**Habits.**—Careful inquiry should be made into the habits and environment. Is the patient in the habit of spending much of his spare time in the open air, or does he spend it indoors chiefly? Is it spent in his own house, in crowded places of public amusement, or in public-houses? Is he in the habit of sleeping with his bedroom window well open or the reverse? What are his habits as regards late hours? What is the history as to alcohol? Where is the house situated, and is it conducted on reasonable hygienic lines?

**Previous Illness.**—An examination should be made into the history of any previous illnesses. Some of these may be of vital importance; others may be ignored altogether.



All previous diseases of the lungs should be specially recorded and a careful note made of pneumonia or pleurisy. The lung which was affected should be determined if possible. Cases of pneumonia which terminate by lysis and have a slow protracted convalescence are, as a general rule, of a tuberculous character. In such cases a prolonged course of treatment on open-air lines should be insisted on until health is completely re-established. For many years afterwards a careful watch should be kept on the general health of such a patient.

With a few exceptions, it may be taken that all pleurisies with effusion are tuberculous. The exceptions to this rule are pleurisy associated with malignant growths, aneurisms, and rheumatism. Unless some definite non-tuberculous cause is found, the pleurisy should be looked upon as being of a tuberculous nature. It is extremely common on investigating a patient's history to note that some years previously there had been an attack of pleurisy, and equally common to ascertain that this most marked danger signal had been altogether ignored, and now it is distressing to find the patient suffering from well-marked pulmonary tuberculosis. On careful questioning he will state that he recovered fairly well from the pleurisy but that he started work too soon and has not been quite the same man since. It should be looked upon as negligence on the part of the medical attendant if he fails to impress on his patient the significance of an attack of pleurisy and the necessity for making, as far as possible, a complete recovery from it. Due warning ought also to be given as to the care of health for many years after the illness. An attack of pleurisy is an indication that there is an enemy in the way which may steadily follow on the patient's track awaiting the first favourable opportunity for attack. It may follow for years without finding such opportunity, and the question as to whether or not such a chance will come depends on the patient's manner of life and the amount of care bestowed on the preservation of health. A golden rule to be remembered by all concerned is, never despise a pleurisy.

**Bronchitis.**—Frequent attacks of bronchitis in young adults must be made the subject of special inquiry, as these

attacks may be due to a tuberculous focus. When a bronchitis is confined to the apex or apices of the lung, this is strong presumptive evidence that the illness is tuberculous in character. Again, where the bronchitis is unilateral, although not necessarily confined to the apex of the lung affected, it should be assumed, until the contrary is proved, that this is a tuberculous manifestation, and a careful examination of the sputum for confirmatory evidence should be made. The author has on several occasions seen such cases diagnosed as simple bronchitis, and tubercle bacilli were afterwards demonstrated in the sputum.

**Previous Tuberculous Disease.**—Due regard must be given to any evidence of previous tuberculous disease. The fact that some evidence of old bone or joint disease is present indicates that the patient is susceptible to attack by some forms of the bacillus. In the case of old cervical adenitis it is not uncommon to find that in later years the patient is attacked by pulmonary tuberculosis in the lung corresponding to the side in which the cervical glands are affected. It has been stated that it is rare for this to happen, but it is more frequent than is generally supposed. Experience of several such cases has given a strong impression that the pulmonary disease in this type of case runs a very much more chronic course, and is distinctly less liable to break down than in some others when the cervical glands have not been involved.

**History of Present Illness.**—One of the first questions asked regarding the present illness is usually, "How long have you been ill?" Often an indefinite statement is made in reply to this. By cross-examination, however, ultimately the approximate date of the beginning of the illness can be made out. This may be a month or a year prior to the patient seeking advice. At other times the reply is, "I was quite well until I took influenza at such and such a time." Careful inquiry may elicit the fact that before the attack of influenza there was a period of slight ill-health, cough, etc., which may have been present many months before the supposed cause. It is important to determine the real as distinguished from the apparent date of onset as nearly as possible, as it gives the physician a more correct idea

of the real duration of the disease. This in turn assists greatly in recognising the activity and rate of progress, and has also an important bearing on the question of prognosis. The physician should never be put off with indefinite statements, but should determine as nearly as possible the approximate date of the beginning of the illness.

**Mode of Onset.**—As a general rule the onset of tuberculosis is extremely insidious. The failure of health is so gradual that it is only when a serious inroad has been made into the health of a patient that he recognises that he is ill. (See Fig. 9.) There may be a gradual failure of health without any catarrhal or other lung signs. The disease may be discovered accidentally in the course of examination for life assurance or, as has happened on many occasions in the last few years, in the course of examination for army service. At other times the striking feature may be anæmia, and no symptoms are present to direct attention to the lungs. Again, dyspeptic symptoms may be the most pronounced. There may be vomiting or gastralgia, with loss of appetite. The consequent loss of flesh is attributed to dyspepsia—the real cause of the symptoms, viz., the lung lesion, being quite ignored. Occasionally the first symptom of the disease is hoarseness of voice, and this as a rule is in the early stages attributed to a cold. Its persistence, however, should direct attention to the chest.

Again, the disease may develop as a remote sequela to other forms of tuberculosis, or to other constitutional diseases which have already been noted. Occasionally the disease first manifests itself by a hæmoptysis, and the alarming nature of this at once directs the attention of both patient and doctor to the lungs. It has been estimated that in about 10 per cent. of cases of pulmonary tuberculosis, hæmoptysis is the initial sign. In one series of 726 cases under the author's care, it appeared as an initial sign in 39, or 5·4 per cent., of the cases.

## CHAPTER VIII

### SYMPTOMATOLOGY

THE early symptoms complained of by the patient are, as a rule, indefinite and vague. There may be some loss of energy, loss of strength, poor appetite, etc. These symptoms, however, are not found in cases of early tuberculosis only, but are met with in other diseases as well. Care should therefore be taken to ascertain if there is any other likely cause for them, before assuming their tuberculous origin. Many patients have been labelled tuberculous, chiefly on the grounds of these indefinite complaints, who in reality are suffering from other causes, such as overwork or lack of holiday. In a greater number, however, the tuberculous nature of the illness has been missed, owing to a lack of appreciation of those very early symptoms.

**Cough.**—There is no case of pulmonary tuberculosis without cough, although, of course, every patient with a cough is not necessarily the subject of lung tubercle, and no cough is pathognomonic of tuberculosis. The presence of a cough which has persisted for some time, say, six weeks, in the case of a patient who has hitherto been healthy, suggests tuberculosis as the cause, and this should be carefully looked for. Often the cough is so slight that the patient denies its presence, but at other times it is a pronounced symptom and directs attention to itself. In early tuberculosis it may be slight and confined to the morning hours, or it may be present both morning and evening. It is rarely troublesome during the hours of the night. At times it is paroxysmal in character. In the later stages of the disease, when the sputum is more abundant, the cough is more frequent, but not necessarily more distressing to the patient. Sometimes, however, it is extremely exhausting, and in

emaciated patients causes involuntary micturition. When the disease has gone on to vomica formation there may be prolonged periods without cough, owing probably to the sputum collecting in the cavity, but with a change of position it gravitates to a more sensitive portion of the lung and an attack of coughing results, with copious expectoration. When the cavity is emptied of its contents the cough ceases again. In some few early cases of disease vomiting associated with slight cough is a prominent sign. This has been termed emetic cough, or, as the French call it, the *toux émétisante*. This vomiting should be carefully distinguished from that which is frequently met with in advanced cases, where it is associated with a cough, violent and paroxysmal. In the cases where the true emetic cough is observed, the patient, half an hour or so after food has been taken, has an irritating feeling at the back of the throat, and with a slight cough the contents of the stomach are ejected, and soon after the patient feels well again. The peculiarity of this cough is that there is no preliminary sense of nausea. In this country a typical case of this distinct and characteristic cough is rarely met with.

A chronic cough must not be taken as a certain sign that the patient suffers from tuberculosis, as it is met with in all other lung diseases. It is found also in chronic cardiac disease where heart failure is beginning to manifest itself; in thoracic aneurism; and in diseases of the larynx and pharynx. In children enlarged bronchial glands are occasionally a source of chronic cough, and in that case the cough may be of the well-known paroxysmal character. It will thus be seen that with so many and so varied probable causes a considerable amount of work has often to be done before the definite cause of the cough can be ascertained.

**Sputum.**—The presence or absence of sputum has to be determined. Oft-times the patient is unaware that sputum exists. In children under five years of age it is extremely uncommon to get expectoration, and it is also unusual to find it in women in the earlier stages of the disease. During an examination a woman may be observed coughing frequently, and obviously swallowing a fair amount of sputum after each cough, while all the time she will



vigorously protest that she has no sputum. Whatever the reason may be, the sputum is much more easily obtainable in men patients. It is probably because men are accustomed to spitting and are less troubled with ideas of delicacy than women. The character and quantity of sputum should be noted, although there is no expectoration characteristic of tuberculosis. In the early stages there may be only a slight amount of glairy mucus, streaked with black, or, it may be, greyish in colour. The quantity may be insufficient to measure with exactness or it may not amount to more than one drachm per day. As the disease advances the sputum increases in quantity, several ounces being expectorated daily, and it is slightly streaked with purulent matter which gives it a yellowish tinge. By and by this purulent matter begins to predominate, and the sputum is then described as muco-purulent. Later it may become altogether purulent. Occasionally it is tinged green, or stained with blood. At other times it is markedly frothy in character, with a small sediment of comparatively solid mucus at the bottom of the sputum cup. As a rule it is odourless, but at times it becomes fœtid. This latter is due to the effect of secondary organisms in the lung cavity. Sometimes a patient complains of the sputum having a sweet sickly taste, but as a general rule there is nothing to attract the patient's attention to the taste of the sputum. When there is a large cavity in the lung the sputum is similar to that from a bronchiectatic cavity—that is, when collected in a glass vessel it is seen to separate into three well-defined layers: a frothy layer on the top, a middle layer of comparatively clear liquid, and a thick sediment at the bottom made up of pus cells, debris, etc.

When sputum is present it should be frequently examined to determine the presence of tubercle bacilli. When the physical signs are uncertain one negative examination is of little importance. Frequent examinations must be made. A good routine procedure in sanatorium practice is to re-examine all the negative sputa every second day during the first month of the patient's residence in the institution. On many occasions it is only after repeated examination that bacilli are found. In one extreme case



under the author's care they were not found until the eighteenth examination. In another suspected case the sputum was examined by a class of twenty-five students, each of whom made a separate film. In one specimen only were the bacilli detected. Such cases will illustrate the amount of weight to be placed on one examination of the sputum where the result is negative. Even if after repeated examination no bacilli are found, the conclusion must not be arrived at that the case is not one of tuberculosis, because in the incipient stage of the disease, the sputum will often be free from bacilli. A sputum containing tubercle bacilli means a breaking-down focus of disease in the lung, and a diagnosis should be made, if possible, before this takes place. The numbers of the tubercle bacilli in the sputum are of no diagnostic value as a rule. Probably all that is implied in cases where they are numerous is that the examiner has been successful in getting a small particle of the sputum teeming with the organisms. If, however, a sputum has been regularly examined, and a gradual diminution of the number of bacilli takes place, then it is reasonable to suppose that this may be of a good prognostic value. If in a suspected case of tuberculosis the sputum is scanty, it may be considered advisable to increase the quantity, so as to obtain specimens for examination purposes, by giving occasional doses (10 grains) of potassium iodide.

**Concentration Methods.**—When the bacilli are so scanty that it is only after several examinations that their presence is detected, in order to facilitate their detection several methods of examination have been introduced. These have been termed concentration or enrichment methods. (1) One simple method is to collect the sputum for twenty-four hours in a conical glass and to incubate this at 37° centigrade for twenty-four hours. At the end of this time the sputum has become more liquid and the solid particles have fallen to the bottom. If the bacilli are present they will be more readily found in a portion of the sputum from the bottom of the glass.

(2) *Antiformin Method.*—Uhlenhuth and Xylander first introduced this method, and the following is a routine way of carrying out the procedure :—

Collect the sputum in a conical jar, and if it is extremely

tenacious dilute with water. Add, from one-quarter to one-half of the volume of diluted sputum, a 50 per cent. solution of antiformin. Stir well and allow to settle for several hours. Sedimentation is assisted by diluting the sputum with an equal quantity of alcohol. After the sedimentation is complete the clear fluid is poured off and the sediment examined. It should be carefully noted that some medium is required to fix the sediment on to a glass slide, and this is usually done by taking some white of egg or a small quantity of the original sputum which is being examined, and mixing it with the sediment. The antiformin dissolves the mucus and any extraneous organisms that are present in the spit, and other acid-fast bacilli are also disintegrated. It is stated that the tubercle bacillus can withstand a 50 per cent. solution of antiformin for three or four days, but the smegma bacillus is dissolved in about half an hour in 15 per cent. solution. A variation of the antiformin method is made by boiling equal parts of sputum and antiformin and then adding an equal quantity of ether. The mixture is then well shaken and centrifuged. A thin white film forms on the top of the liquid, and if tubercle bacilli are present they will be found in this film.

**Inoculation Test.**—Occasionally it is desired to test the sputum in a still more delicate fashion, and this is done by inoculating a small portion into the leg or abdomen of a guinea-pig. These animals are so susceptible to tubercle bacilli that if there are any in the sputum their presence will be shown in a definite time, and the manifestations follow in a regular order. In from three to four weeks the glands in the neighbourhood of the site of inoculation become enlarged. Then the internal organs, lungs, spleen, liver, become involved, and later the animal dies of general tuberculosis. At the end of six weeks or two months, however, the animal is usually killed (instead of being allowed to die of the disease), and a careful examination is made for the evidence of the presence of tuberculosis.

Various efforts have been made, but without much success, to shorten the period between inoculation and the manifestation of well-marked symptoms of the disease. One method that has been tried in order to hasten the growth of

the bacilli was bruising in a clamp those lymph glands which are near the site of inoculation, in the hope that growth will take place more quickly in a damaged gland. Other observers have exposed the animal to massive doses of X-rays. Others again, a few days after inoculation, have performed one or other of the cutaneous tests on the guinea-pig to see if the animal will react. In some cases a definite skin reaction has been observed within ten days of inoculation. In the majority of instances these attempts at an early diagnosis have failed entirely, and there seems little to be gained in any individual case by such procedure.

Much work has been done on the cytology of the sputum, but so far nothing of importance has resulted from these studies. Elastic fibres may be looked for, and with a little trouble they are found when the lung is breaking down.

A simple method of examining for these fibres is to pick out a solid piece of sputum, press it between two glass slides, and examine by a low-power microscope; if the fibres are present they are then easily detected. Another method of examination is by boiling the sputum with an equal quantity of a 10 per cent. solution of caustic potash. After boiling dilute with water, centrifugalise, and examine the sediment. Elastic fibres are rarely searched for now. The routine examination for the bacilli and their significance when found, has robbed the elastic tissue of its importance from a diagnostic point of view. Albumen is often found in the sputum of consumptive patients, and has been thought to be of diagnostic importance. This point will be further dealt with under the heading of Special Tests.

**Hæmoptysis.**—From a consideration of the pathological processes which take place in pulmonary tuberculosis, it is surprising that any case of the disease should run to a fatal issue without several attacks of hæmorrhage, and yet it is a well-known fact that many cases terminate fatally without any hæmoptysis. The frequency with which this accident occurs varies, but it has been estimated to take place in from 60 to 70 per cent. of the cases. In a series of 726 cases admitted to the sanatorium under the author's care, it had occurred in 46 per cent. of the cases, and these were all in the earlier stages of the disease. That it is not more

common is explained by the slow advance of the disease permitting thrombosis to take place in the small vessels before they ulcerate.

*Cause.*—In many cases no definite cause for the hæmorrhage can be ascertained, but at times a patient will say that he had been coughing violently, lifting some weight, or performing some physical exercise. At other times the patient will attach the blame to some momentary excitement which he has just undergone. As a general rule no warning is given before the hæmoptysis takes place, and a great number of the attacks occur during sleep, or in the early morning before the patient rises. It is not clear why this should be so, as there is no special physical strain at that time. Patients will sometimes state that they have had a very definite premonition as to hæmoptysis, complaining of tightness and discomfort in the chest—this premonitory feeling lasting several hours before the hæmorrhage occurred, and always being relieved by the bleeding. Sex has very little influence in the later stages of life, but in middle life hæmoptysis appears to be more common in males than in females. It is rare at both extremes of life, and is especially rare in childhood, although at times a copious bleeding may take place in quite young children. A considerable number of observations have been made on the relationship of barometric pressure to the occurrence of hæmoptysis, but the observations failed to show that there was any definite connection whatever; yet in sanatorium practice it is not uncommon to have two or three cases of hæmorrhage occurring about the same time.

The immediate causes of the hæmorrhage vary. In slight cases it may be due to a hyperæmia of the inflamed area of the lung where blood-stained serum oozes through the epithelium. In advanced cases, with cavity formation, slight hæmorrhage may be produced by the oozing of blood from the soft granulation tissue lining the cavity, just as one may see a sanguineous discharge from an old varicose ulcer. The larger hæmorrhages are due to the erosion of some small blood-vessel which is ruptured before thrombosis can take place, the tuberculous ulceration having destroyed the supporting tissues of the vessel with the external coat of the

artery itself. In other cases this loss of support leads to the formation of small aneurisms of the pulmonary arteries, and the rupture of these will at times cause copious hæmoptysis.

*The Relation to the Stage of the Disease.*—Speaking generally, only slight hæmorrhages take place in the early stage of the disease, and the more profuse bleedings in the later stages. Hæmorrhage may be found occasionally in persons who are apparently in perfect health. Most physicians of any length of experience have met with patients who in youth had a definite hæmoptysis but who never showed, in after years, the slightest evidence that there was any tuberculous disease present. The hæmorrhage occurs while the individual appears to be in perfect health, and after the hæmorrhage he appears to remain in perfect health. There is no doubt that in the great majority of these cases the bleeding is due to a genuine tuberculous ulceration in the lung, not large enough to produce any apparent general systemic disturbance, but sufficiently large to erode one of the blood-vessels. In other cases the hæmorrhage, though very slight in amount, directs attention to the lung, and manifestations of the disease are quite apparent on examination. In advanced cases the hæmorrhage tends to be more abundant. Several pints may be expectorated during one attack. Certain terms have been employed to describe, for practical purposes, the amount of bleeding that has taken place at one time. For instance:—

Coloured sputum, when the sputum is merely stained.			
Small hæmorrhage, when the quantity is less than 1 ounce.			
Moderate	„	„	from 1 to 4 ounces.
Profuse	„	„	from 4 to 10 „
Copious	„	„	more than 10 „

In advanced cases of the disease it is rare that death is caused by hæmoptysis. Probably not more than 2 per cent. of the cases die from this complication. Occasionally a patient may be met who suffers from repeated hæmoptysis, so that for a prolonged period, extending over several years, there are recurring attacks of hæmoptysis. To such cases the term of hæmorrhagic phthisis has sometimes been given.



*Results.*—While hæmoptysis rarely terminates in death, it gives rise to a considerable amount of anxiety to the patient and his friends. When the hæmorrhage is profuse the patient lies with pale, anxious-looking face, blanched lips, restless eyes, and quick pulse.

The fear of increasing the amount of hæmorrhage keeps him almost motionless, except when the irritation of the blood in the bronchial tubes compels him to cough and turn his head to expectorate. The excited patient, his excited friends, and the presence of blood-stains on the bed-clothes and on the side of the sputum mug, make a picture that is alarming and not easily forgotten. In the cases where death occurs through the rupture of a large vessel or aneurism, the fatal result takes place with extreme rapidity. Indeed, there is nothing more tragic in the whole of my experience than these sudden deaths. The patient may leave his room to take a morning walk in apparently perfect health, being well nourished and usually sunburnt, when a hæmorrhage may occur of such a profuse character that within a couple of minutes he is dead.

When a patient recovers from the initial attack of hæmorrhage there are some risks that he is exposed to:—(1) There is a chance that the blood infected with tuberculous material may flow into other (healthy) portions of the lung and infect these areas with additional foci of disease. Fears on this score are more theoretic than practical, as it is very seldom that a hæmorrhage is followed by anything approaching a generalised distribution of the disease. (2) Another danger is that the blood may so irritate the lung as to set up a broncho-pneumonia. This happens in a few instances, but it is by no means common.

Often a hæmorrhage takes place in an acute febrile case where the tuberculous process is rapidly spreading and the lung disintegrating, and, in these instances, the patient may rapidly succumb to the disease, not owing to any irritation produced by actual extravasation of blood, but to the exhaustion produced by its loss. Relatively few patients become permanently worse after a hæmoptysis if they have been in a comparatively good condition of health prior to the bleeding.



**Hæmoptysis not due to Tuberculosis.**—In many cases of apparent hæmoptysis the bleeding is not due to any tuberculous process in the lung, and it should always be carefully ascertained, if possible, what the precise origin of the bleeding is. The term spurious hæmoptysis has been applied to bleeding not due to lung disease. The following causes may be instanced.

*Disease of Mouth and Nasopharynx.*—In many patients coloured or blood-stained sputum is due to adenoids and enlarged tonsils. In some children and adolescents who suffer from these diseases, there is frequently found in the mornings a blood-like stain on the pillow, and this has on several occasions given rise to a diagnosis of tuberculosis. In other instances, the child suffers from spongy gums, and this condition also often causes a slight amount of bleeding which is at times mistaken for a genuine hæmoptysis. Again, in cases of chronic pharyngitis the sputum will often be stained with blood.

Associated with enlarged tonsils there is often a considerable amount of enlargement of the adenoid tissue at the root of the tongue which has been termed the lingual tonsil. On laryngoscopic examination it may frequently be noted that coursing over the surface of this adenoid tissue is a network of small delicate veins or capillaries. These having little supporting tissue rupture easily, and cases of slight hæmorrhage occur in which the bleeding can be traced to this source.

*Diseases of Lungs.*—In acute bronchitis, during a paroxysm of coughing, slight hæmoptysis is not uncommon. In chronic bronchitis and emphysema it is also not infrequently seen, especially if the patient suffers from definite arterial degenerative changes. This fact should be carefully remembered, as there is a tendency in all cases of emphysema, when a hæmorrhage occurs, to assume at once that it is due to a focus of tuberculosis. Hæmorrhage also takes place in the early stages of pneumonia, in cases of bronchiectasis, malignant growths, gangrene of the lung, and in hydatids and actinomycosis.

*Cardiac Disease.*—In valvular disease of the heart, especially mitral stenosis, there is often found recurrent

and profuse hæmoptysis. One of the frequent mistakes made is to neglect examination of the cardiac condition and to assume that such a profuse hæmorrhage can only be due to a tuberculous lesion. This has happened repeatedly, and had the heart been carefully examined, there would have been found unmistakable evidence of the stenosis.

Hæmorrhage also takes place in mitral regurgitation, particularly when the left side of the heart is becoming dilated and a condition of venous congestion created in the lungs. In these valvular conditions crepitations are often found at one or other apex, and these crepitations tend to confirm the examiner in his erroneous idea that he is dealing with a case of pulmonary apical tuberculosis. Before making a diagnosis of tuberculosis where a well-marked cardiac lesion exists, it is a good rule to insist on the presence of tubercle bacilli in the sputum.

In cases of pulmonary infarction there is also a considerable amount of hæmorrhage, but here it is usually associated with sudden pain in the side and often with pleuritic friction. In aneurismal conditions there is sometimes prolonged slight staining of the sputum before any definite bleeding takes place. This is due to the slow oozing of blood through the wall of the aneurism, and is often a precursor of a large fatal hæmorrhage.

*General Diseases.*—In some general diseases such as purpura, pernicious -anæmia, and the specific fevers, it is not uncommon to have some hæmoptysis, and this quite independent of tubercle of the lung. In the last great epidemic of influenza it was a comparatively frequent occurrence for a patient to have a decided hæmoptysis, especially if the influenza was complicated by the typical pneumonia which was then so common. In hysterical subjects hæmoptysis is often complained of. In some instances patients may actually injure the gums wilfully, or produce hæmorrhage from them by prolonged or forcible sucking, so as to create the impression that they suffer from serious disease. Several cases have come under my own observation where patients have been sent to, and have been treated in, a sanatorium for considerable periods with no other sign of tuberculosis than this spurious hæmoptysis.

**Differential Diagnosis of Hæmoptysis.**—In the coloured sputum which is found in some cases of adenoids, enlarged tonsils, and spongy gums, the colour of the blood is distinctive. It has a brownish, rusty-looking appearance, quite different from that of pure blood, and once seen there is little chance of mistaking it. When the adenoids and tonsils are attended to, the hæmorrhage ceases. In the spurious hæmoptysis of neurotic women the general behaviour of the patient is of importance, as other manifestations of hysteria are likely to be shown. Careful examination of the buccal mucous membrane may reveal evidence of wilful injury to the gums. It often requires much acute observation on the part of the medical and nursing staffs to detect these cases, but, if the necessary care is taken, this can usually be accomplished.

In the hæmoptysis which occurs occasionally in acute and chronic bronchitis it is, at times, quite impossible at the outset to exclude the possibility of a small tuberculous focus being the cause. This can only be accomplished by a careful study of the patient over a prolonged period. In bronchiectatic conditions, where there is hæmoptysis, it is, again, often impossible to exclude tuberculosis as the cause of it. If the history of the case is familiar, and it is known that bronchiectasis is present, this will help considerably; but if the patient is seen for the first time after the hæmoptysis, it is then extremely difficult to assign the real cause. The general rule as to the situation of the disease is of much assistance, bronchiectatic lesions being usually found at the base and tuberculous conditions being most frequent at the apices. The patient would, however, require to be under observation for some time before a definite opinion could be given with any degree of accuracy.

When hæmoptysis occurs in cardiac disease the presence of the heart lesion is of prime importance in settling the origin of the bleeding. Tuberculosis is rarely found in patients who suffer from mitral stenosis, and, as already indicated, where a well-defined heart lesion is present it is a sound rule to insist on the presence of tubercle bacilli in the sputum before making a diagnosis of tuberculosis. This is all the more important as infarctions and local œdemas

give rise to physical signs in the chest similar to those caused by tuberculosis.

The distinction between hæmoptysis and hæmatemesis is as a rule quite easy, but at times it is extremely difficult to decide as to the origin of the bleeding, especially when dealing with ignorant or non-observant patients.

In hæmoptysis the sputum is usually frothy and mixed intimately with mucus, while in hæmatemesis it is not frothy. In hæmoptysis it is alkaline in reaction, and in the other acid. A point of particular importance is that in hæmoptysis, after the acute hæmorrhage has ceased, it is quite a usual thing for the patient to expectorate small dark-coloured clots of blood for a day or two, whereas this never occurs in hæmatemesis. Again, the previous history of the patient is of importance in helping to decide this difficult question. In some instances there is a definite history pointing to gastric disturbance, with dark-coloured stools; in others the indications point to pulmonary mischief. Much difficulty is often experienced in those cases in which the patient unwittingly swallows blood which has come from the lung and vomits it later. Even when the greatest care is taken, there is always a considerable number of cases where it is almost impossible to decide whether the bleeding has been from the lungs or from the stomach. The patients themselves are not of much assistance, as they are not sufficiently careful in observation to make their statements of value.

**Fever.**—A careful study of the temperature of the patient is of vital importance in cases of early tuberculosis, as it is one of the most valuable single signs that we have. It should be accepted as a rule that there are *no cases of active tuberculosis without some disturbance of the temperature*. Conversely, it may be said that if there is no fever there is no active phthisis. This simple test should be carried out much more carefully than is commonly done, and more reliance placed on it.

The only exceptions that might be made to this rule are cases of chronic fibroid phthisis or of tuberculosis of the aged, associated with emphysema. Here the disease may progress slowly for a term of years without any definite fever being

detected. These, however, are in a quite different class from early tuberculosis, and it is likely that, if extreme care were taken, it would be found, even in these fibroid cases, that fever was not infrequently present.

In early tuberculosis we do not expect gross disturbances of the temperature. We are dealing with slight perturbations which may only be  $1^{\circ}$  or  $1\frac{1}{2}^{\circ}$  F. above the normal. Hence there is urgent necessity for great care being taken in the application of thermometric methods.

*Technic.*—The first essential is to make sure that the thermometer registers accurately. When acting as Superintendent of a Sanatorium the author made it a routine practice that all thermometers given out for use in the wards were corrected against a standard thermometer, and it was interesting to note the variations that existed in some of the instruments. They might vary from  $1^{\circ}$  to  $1.5^{\circ}$  or even  $2^{\circ}$  F. from a thermometer which had been corrected at Kew.

There are three methods by which the temperature is usually taken.

*Axillary Temperature.*—This is commonly employed in hospital wards, and in cases of high fever it is useful, but for the purpose of recording the minute changes which take place in early pulmonary tuberculosis it is quite useless and should never be used.

*Oral Temperature.*—When proper precautions are taken, this method gives a satisfactory record of the temperature. The following are the main precautions which should be observed. Nothing cold or hot, either liquid or solid, should be taken for, say, thirty minutes before the temperature is being recorded. The mouth should be closed for this period, and no speaking or mouth-breathing permitted. The face of the patient should not be exposed to cold winds, as it has been found that such exposure will interfere gravely with the temperature record. Little reliance can be placed on any thermometric observations in the mouth when the thermometer has been placed there for less than five minutes. That period of time should be the minimum, and in cases of doubt it should be extended to ten minutes. Often a patient's temperature, taken with a one-minute thermometer, may show at the end of that time no elevation whatever, but



if kept in for five minutes the thermometer may show a moderate degree of fever.

The bulb of the thermometer should be placed under the tongue and the lips gently closed over the stem. The oral method when employed in children is somewhat expensive, as the children are extremely apt to bite through the stem of the thermometer.

*Rectal Temperature.*—This gives the most accurate results in thermometry. There are æsthetic reasons why it may not be universally employed, but in sanatoria it is used regularly for both sexes and without any grave objections. Care should be taken to see that the bulb of the thermometer is introduced into the rectum, and not, as sometimes happens, grasped by the external sphincter. It should be left *in situ*

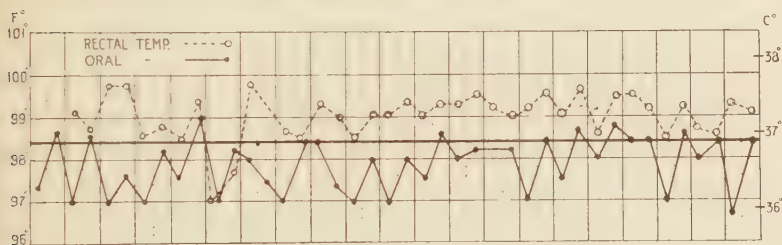


FIG. 10.—Oral and Rectal Temperatures, taken simultaneously.

for at least two minutes. As a modification of this method, it might be mentioned that nearly equally satisfactory results are obtained by the passage of urine over the bulb of the thermometer, provided that there is an adequate quantity of urine available.

In comparing the results of these methods the rectal temperature is by far the most satisfactory, but if it is remembered that the oral temperature varies from  $0.5^{\circ}$  to  $1.0^{\circ}$  F. below the rectal, it might be taken as quite sufficient for all practical purposes. In these cases, however, where the question of a diagnosis is concerned, the rectal temperature is to be preferred. The underlying chart (Fig. 11) will show at a glance the difference between the rectal and the oral temperatures.

*Types of Temperature.*—In considering the temperature in tuberculosis, it is important to settle what constitutes a

normal temperature. The author has had observations made on men who have to all intent appeared normally healthy individuals, and the temperature for a period of two weeks ranged from  $97.8^{\circ}\text{F}$ . in the morning to  $98.8^{\circ}\text{F}$ . in the evening.

In order, however, to allow for slight individual variations, it may be taken that a normal temperature may range from  $97.8^{\circ}$  to  $99^{\circ}\text{F}$ . In the healthy individual it rises after a full

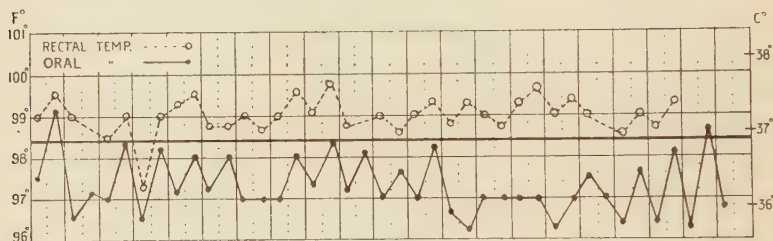


FIG. 11.—Chart in Early Case without physical signs.  
Tubercle bacilli were found later.

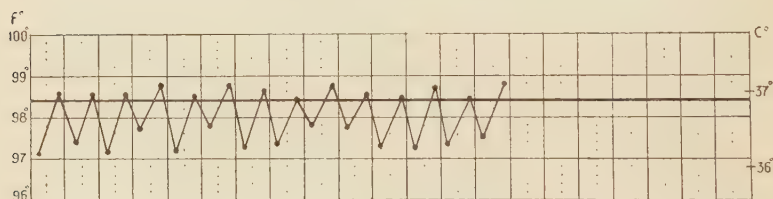


FIG. 12.—Rectal Temperature in a healthy man.

meal, after exercise, or after any degree of excitement. In the daily variation the maximum temperature occurs from about 3 P.M. to 6 P.M., so that in some sanatoria it is customary to take the evening temperature about 4.30 P.M.

*Temperature in Early Cases.*—The diagnostic value of the temperature in early cases of the disease has already been insisted upon. The author has long since come to the conclusion that the fact of an adult patient's temperature rising frequently above  $99.2^{\circ}\text{F}$ . in the evening, especially if associated with a subnormal temperature in the morning, is, in the absence of other causes of fever, wellnigh pathognomonic of early tuberculosis. So much importance is

attached by him to this type of fever, that given a case with only slight cough or slight constitutional disturbance, with few or no physical signs in the chest, but with the variations in temperature already referred to, he should unhesitatingly diagnose early active pulmonary tuberculosis. In any

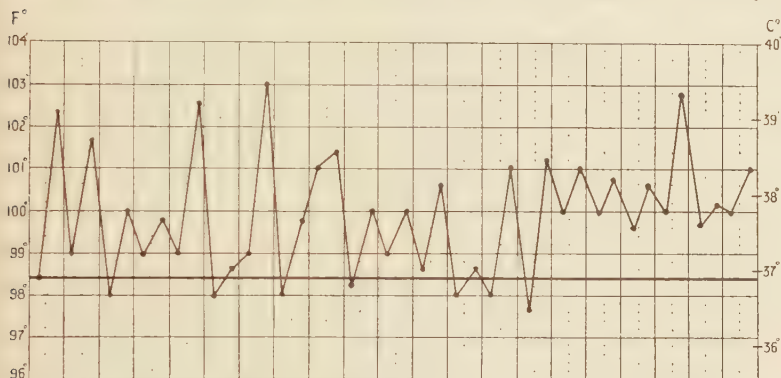


FIG. 13.—Intermittent Temperature in advanced tuberculosis.

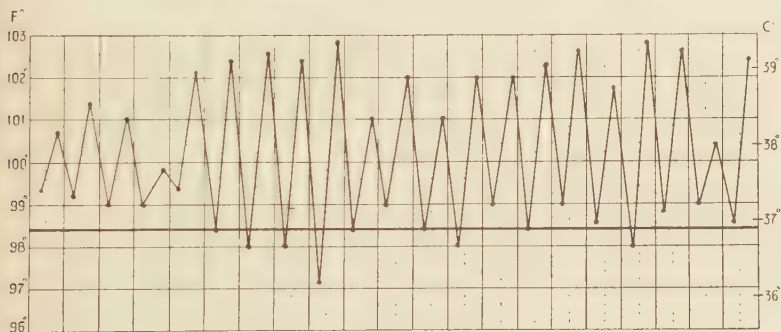


FIG. 14.—Intermittent Temperature in advanced tuberculosis.

suspected case which is under observation, either in the patient's own home or in a sanatorium, it is advisable that the temperature should be taken every two hours from, say, 7 A.M. until about 10 P.M. If these observations are carried out for a week or ten days there is little likelihood of missing any transient fever should such occur. Afterwards the temperature might be taken twice daily, say at about 7.30 or 8 A.M. and about 4 P.M. Any intelligent patient, with

a little education, can easily perform this task himself. In some neurotic individuals, however, it is advisable that they should not be made too familiar with temperature changes, as the very fact that they are anticipating an elevation of temperature seems quite sufficient to produce one.

In such a patient a prolonged attack of evening fever has been cured by forbidding the taking of the temperature, and by stating that there was no fever. It should be remembered, however, that certain conditions other than tuberculosis give rise to a slight evening pyrexia. Where there is any chronic suppurative process present, as for instance in the nasal sinuses, in bronchitis, bronchiectasis, and in lung tumours, there is often evening pyrexia.

In advanced cases of tuberculosis where the process is active, any type of fever may be met with, and it should be noted that there is no pathognomonic variety. Intermittent fever may be seen with morning remissions to subnormal levels and an evening rise, it may be, to  $103^{\circ}$  F. or so. Again, it may be a continuous temperature ranging without intermission between  $100^{\circ}$  and  $103^{\circ}$  or  $104^{\circ}$  F. Such temperatures as these are of extremely bad augury. If these temperatures last for a few weeks and resist all attempts at lowering, then a fatal issue may sooner or later be expected. Occasionally an inverse temperature is met with when the rise is in the morning and the fall in the evening.

**Loss of Weight.**—This is a well-recognised sign of tuberculosis, and, in the absence of other causes to which the loss may be attributed, it is an important sign. Loss of weight, in the first instance, is due to the wasting of adipose tissue. In the later stages, when the disease is making serious inroads, not only is there loss of adipose tissue but the muscular tissue is atrophied. This is also seen in the muscles which clothe the upper portion of the chest walls, viz., the pectoral, supra-spinatus, trapezius, and sterno-mastoid muscles. This wasting gives a characteristic appearance to the tuberculous patient: the sunken cheeks, the emaciated chest with the ribs standing out prominently, and the hollowing above the clavicles and over the supra-spinous areas. In later stages there is wasting of the muscles of the limbs which produces in them marked

weakness. The causes of the loss of weight are various. It may be due to the direct result of the toxæmia, or to the anorexia which is such a common feature, or to actual dyspeptic conditions.

The loss or gain of weight when the patient is under treatment is of some considerable prognostic value. As a rule a gain in weight is evidence of successful resistance to the disease, and consequently makes the outlook somewhat brighter. It has to be confessed, however, that in many patients, after admission to a sanatorium, there is a distinct progressive gain in weight, and yet all the time the disease in the lung is advancing, and after a few months the patient begins again slowly to lose the weight he had gained in the earlier stages. Progressive loss in weight is of bad prognostic significance. It should be noted that there are some patients with active progressive lung lesions who are stout and well nourished. Any physician of experience must have met this type of individual who rather resents the idea that there is any disease in his lungs, and yet, in spite of the adipose tissue, the disease progresses.

The loss of weight in early tuberculosis is, as a rule, not rapid. It may only amount to one or two pounds per month. It is the persistence of the loss rather than the amount which is the important point. If an adult should, over a period of six months, lose weight steadily to the extent of six pounds there would be good ground for suspecting tuberculosis. In some cases in which the physical signs may indicate early disease, rapid and extreme emaciation is met with. This should be taken as indicative of an acute or extensive pulmonary lesion even in the absence of much apparent lung disturbance.

**Cardio-vascular Symptoms.**—Very early in the disease, and often before any definite lung lesion can be detected, tachycardia may be noted. This is a sign of much importance. It is present in the great majority of cases where there is activity in the lung process. A pulse rate of 90 to 100 is quite common in cases which are apparently afebrile. With a slight degree of fever, the rate may rise to 120 per minute. Often the tachycardia will persist for a prolonged period even after all local evidence of pulmonary



activity has passed away. Such a persistence should always make one doubt the permanence of the arrest of the disease. Tachycardia which does not improve with rest is a sign of grave import for the future of the patient. It is difficult to give a satisfactory explanation of the cause of tachycardia. Many theories have been advanced, such as neuritis of the vagus, myocarditis, undue smallness of the heart, and tuberculous toxæmia. The latter is probably the most general cause, as the toxin is apparently a neuro-muscular poison.

*Palpitation* is another symptom not infrequently found in tuberculosis. It may be met with in early cases, especially when associated with anæmia. It is also seen in late cases, and particularly in those where the heart is displaced because of extra-cardial fibrous contraction. In all cases of palpitation and tachycardia, where the cause is not clear, the lungs ought to be examined for evidence of tuberculosis.

*The blood pressure* is frequently low in early cases of the disease, and is almost always so in progressive and later stages. It may vary from 80 to 120 mm. So in any suspected case of early tuberculosis a low blood-pressure would be of some value in forming a diagnosis.

*The heart* itself shows no definite change in early tuberculosis except that in a great many patients it is slightly smaller than usual, and more vertical. This is the so-called pendant heart, which is well seen on X-ray examination. This condition is probably due to lack of muscular development, owing to a disinclination for exertion which is so often found in these patients.

In the blood there are no special characteristic changes. Slight anæmia is often found. Diminution of the hæmoglobin is frequent, and under sanatorium regime its increase is one of the first decided improvements noted. The percentage of hæmoglobin may rise in a few weeks from 60 per cent. to 90 per cent. There is no definite alteration in the number of the cells. An occasional leucocytosis may be detected, but there is nothing diagnostic in it. Arneth's differential count is not of any practical value in diagnosis.

**Gastro-intestinal Symptoms.** — Amongst the early symptoms in tuberculosis are to be found some referable

to the gastro-intestinal tract. It has been said that all consumptives are dyspeptic, and it is true that dyspeptic symptoms are extremely common. One of the earliest subjective symptoms is anorexia. Many consumptive patients have all their lives been known as "small eaters." They have apparently been living on a bare "subsistence allowance." As the disease advances the anorexia becomes more marked and a positive distaste for food may develop. Indeed, one of the common statements from patients or their friends is that if only the appetite could be increased everything would soon be well. Loss of appetite is often most pronounced in the morning, and the patient rarely partakes of anything in the nature of breakfast. This anorexia is in the main of toxic origin, as no pathological alteration can be detected in the stomach to account for it. Fishberg points out that the anorexia of tuberculosis is independent of fever. As a rule, in other diseases loss of appetite is in definite relation to the temperature, and with a high fever anorexia is complete. In tuberculosis, however, a patient with a considerable degree of evening fever may enjoy a hearty meal. No constant pathological change can be found in the secretory function of the stomach. In some cases there is hyperchlorhydria; in others, hypochlorhydria.

In advanced cases dilatation of the stomach is a common feature, and this was especially so in the days when overeating, or superalimentation as it was called, was enforced in the treatment of the disease. Fermentive dyspepsia is not uncommon, and is due to disturbance of the proper secretion of gastric juice and to motor insufficiency. This latter arises from the general loss of muscle-power, common in advanced stages of the disease.

A catarrhal condition of the gastric mucous membrane is frequently found in late stages of the disease, giving rise to pain after food and a sense of tenderness or discomfort in the epigastrium. Actual tuberculous ulceration of the stomach is rare. In the far advanced stage of the disease the tongue is red and raw-looking, and often there are aphthous patches on the buccal mucous membrane. Some writers have claimed that constipation is a symptom of import-

ance, others again have stated that diarrhœa is more commonly met with; but neither of these symptoms are of any value from a diagnostic point of view in the early stages.

In the later stages diarrhœa is a prominent symptom. It may be due to a catarrhal condition of the intestinal wall, caused in some measure by the swallowing of expectoration. In other cases it is due to definite tuberculous ulceration of the bowel, and in rare cases it may be caused by amyloid changes. When there is definite ulceration there are often symptoms of pain in the abdomen some time after taking food, with tenderness on pressure. The diarrhœa is intractable, and the stools often fœtid and contain pus or blood. Perforation is an infrequent accident. Occasionally after ulceration an obstinate and increasing constipation comes on as the result of cicatricial contraction of the bowel wall. This contraction ultimately leads to great emaciation of the patient, and this with hypertrophy of the bowel above the constriction enables the peristaltic action of the bowel to be detected through the abdominal wall.

**Integumentary Symptoms.**—Amongst the symptoms of tuberculosis, one of the best known is *night sweating*. This condition is often extremely marked, and adds a distressing feature to the case. The night sweating of tuberculosis is frequently confused in children with that which takes place in rickets. Indeed, many cases of rickets have been diagnosed as tuberculosis chiefly because of this symptom. It should be remembered, however, that the sweating of tuberculosis comes on in the early morning—round about 3 A.M.—while the sweating of rickets comes on soon after the patient falls asleep, and is confined chiefly to the head and upper part of the chest. In some cases of tuberculosis the perspiration is slight, but in others it is so profuse as to drench the patient's sleeping apparel. Amongst advanced cases there will be met, at times, patients whose clothes have to be changed once or twice during the night because of the discomfort. The cause of this sweating is a little obscure. Naturally one associates it in some way with the toxins of the disease, which stimulate overaction of the secretory glands. This may be an attempt on the part of

nature to throw off these toxins, circulating in the blood, through the sweat glands. Another explanation which is brought forward is that the perspiration is due to exhaustion, and that during the early hours of the morning, when vital processes are at the lowest, the blood-vessels supplying these sweat glands undergo dilatation, with the result that there is an excessive secretion of sweat. This exhaustion theory has some weight added to it by the fact that, often just before the patient goes to sleep, if a glass of hot milk or some other nourishing fluid is given, the night sweat, which would otherwise have occurred, is warded off. Night sweats are not often seen in patients in a sanatorium nor when the bedroom is well ventilated, and, certainly, the "colliquative sweating" of the old writers is a thing of the past in a well-conducted institution.

*Pubescent Skin.*—An old, time-honoured observation amongst the laity is that children with soft downy hair in the interscapular region are prone to tuberculosis. There is some little amount of truth in this observation, but of course all such children do not necessarily become tuberculous. This downy hair is commonly found in weak, ill-nourished, or delicate children, just the type who are apt to form the victims of this disease. Indeed it is quite probable that in some instances this delicate appearance of the child is the outcome of tubercle already implanted in the system.

*Myotatic Irritability.*—This is a symptom often seen in tuberculosis. It cannot be said to be pathognomonic of the disease, but its occurrence is of sufficient frequency to demand attention. It is best seen in the pectoral muscles, and can be brought out well by forcibly flicking the muscles with the fingers, when a wave-like contraction takes place in the underlying fibres. Some importance should be attached to it in making a diagnosis when other signs and symptoms are vague, but it must be remembered that this irritability is seen in many other wasting diseases. With myotatic irritability there is often also observed myoidema, *i.e.*, a local temporary contraction of muscle producing a slight evanescent cutaneous swelling. Occasionally this irritable condition of the skin and pectoral muscles is a pronounced feature and attracts the physician's attention to the chest.

In a case recently under my care, this was the only feature which had attracted the doctor's attention. The patient had a slight cold apparently, but was supposed to be in a healthy condition otherwise, and capable of taking part in athletic games, so it had been deemed unnecessary to take any special notice of the chest. He was sent with a note drawing attention to the myotatic irritability. On examination it was discovered that the youth had a well-marked cavity in the base of one lung. Sometimes the myotatic irritability is more marked on one side of the chest than on the other, the side corresponding to the lung lesion being chiefly affected.

In the later stages of the disease there is often seen pronounced curving of the finger-nails, with or without clubbing of the fingers. In some cases the clubbing is very evident, and may amount to genuine pulmonary osteoarthropathy, the bony as well as the soft structure of the fingers being involved. Cases are met with where the bones forming the ankle and wrist joints are affected, but the best examples of this are found in bronchiectasis rather than in tuberculosis.

**Signs referable to Genito-urinary System.**—In early cases of the disease there are no changes in the urine worthy of note. Albuminuria is not more common than in the average population. In advanced cases, however, albuminuria is found more frequently. When present it does not necessarily mean that the kidney is the seat of tuberculous disease, as there are other causes. It may be due to a simple nephritis, to the effects of fever, or to a definite tuberculous deposit in the organ. In far advanced cases of the disease, it may be due to amyloid disease. When the kidney is the seat of a tuberculous process there may be no localising symptoms to be noted. At other times there may be dull aching pain in the back, with frequent micturition. Blood may be present in the urine in varying amount. Welsh, on examining the kidneys of persons who had died of tuberculosis, found tubercle present in about 53 per cent., but other observers have found a much smaller incidence. Towards the end of life if œdema sets in, albumen may then be detected in the urine, but this is due to passive congestion.



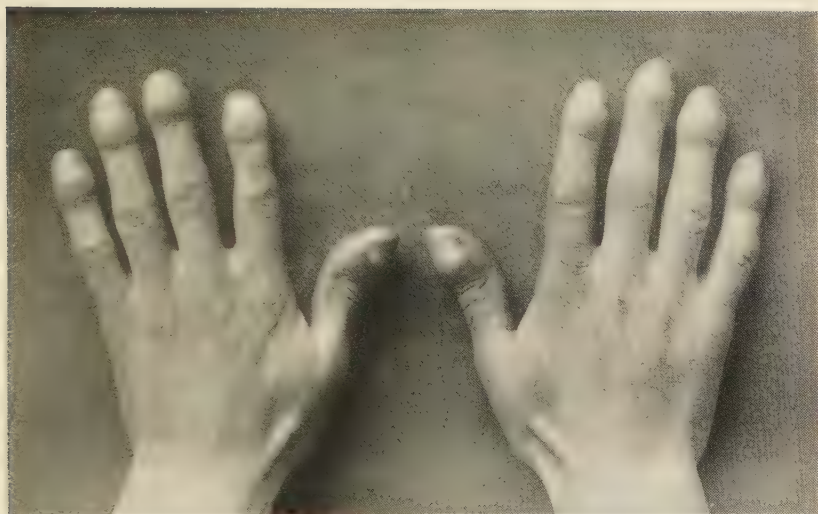


FIG. 15. Hands showing Club Fingers.



FIG. 16.—X-Ray of above.

[Face page 86.



In the early stage of the disease no definite alteration can be detected in the mineral content of the urine, but, in later stages, it is stated by some French authorities that there is a diminution of the phosphatic and calcium salts.

In advanced cases Ehrlich's diazo reaction is usually present. This test is performed as follows:—

1. A saturated solution of sulphanilic acid in a 5 per cent. solution of hydrochloric acid.

2. A 5 per cent. solution of sodium nitrite.

3. A saturated solution of ammonia.

Take a few c.c. of urine in a test-tube, add an equal quantity of solution 1, then a few drops of the sodium nitrite solution. Shake until a froth forms, add ammonia (1 c.c.) until the mixture is alkaline, when the liquid becomes of a port-wine colour, *with the froth red* if the test is positive. This test was at one time thought to be of much prognostic value, but it does not now occupy a place of much importance.

Amongst the symptoms referable to the reproductive organs, disorders of menstruation are the most common. *Amenorrhœa* is frequent, and especially so in girls in whom menstruation has only been recently established—the disappearance of the discharge being looked on as the cause of the ill-health instead of, as it really is, the effect. In all cases of amenorrhœa coming under observation for the first time a careful examination for lung disease should be made, as thereby many mistakes will be avoided.

At times dysmenorrhœa is noted: at other times menorrhagia: but these are of no special significance. Conception is not interfered with by tuberculosis of the lungs. Indeed some writers have expressed the view that tuberculous women are more prolific than the healthy. Pregnancy has long been thought to have a disastrous effect on the tuberculous woman. The famous aphorism of Dubois has generally been accepted in this country—"When a woman who has phthisis marries she may survive her first confinement, a second with great risk, a third never." The disease makes but apparently little progress during the pregnancy, but after delivery it flares up and advances often with great rapidity.

**Symptoms referable to the Nervous System.**—One of the lessons taught us, in this country at any rate, by the war, was that many of our neurasthenic patients were suffering really from pulmonary tuberculosis. In not a few who were so labelled, and who had all the clinical symptoms of neurasthenia, such as sleeplessness, indefinite pains in chest, tachycardia, palpitation, irritability of temper, headaches, languor, etc., an examination of the chest revealed active tuberculosis. It may be assumed that all the symptoms named are due to a tuberculous toxæmia. Unilateral flushing of the cheek is seen at times—the flush being in the same side as the affected lung. Unilateral sweating of the face is occasionally seen but it is very rare. Inequality of the pupils if looked for is often found—a dilatation of the pupil on the affected side being most frequently noted. It is met with in apical lesions with pleurisy, the latter causing irritation of the cervical sympathetic. Hyperæsthesia of the skin over the affected area has been observed. It is commonly due to underlying pleurisy. Some have stated that it is not uncommon in the interscapular area, but this the writer has not noted.

*Psychic Changes.*—The *spes phthisica* is the most noticeable mental feature of the advanced consumptive. He will hope to the last. A dying man within a few hours of death will declare that if only his cough could be cured he would be well, as, except for it, he feels fit for anything. This seems to be one of the compensations of the disease. Certain it is that this hopefulness is not found in any other chronic disease. No one has met a hopeful patient dying of cancer, and yet a dying consumptive may be found discussing his marriage plans. When massed together in institutions, consumptives are difficult to manage and require great tact on the part of those in charge. In fact, the value of a sanatorium depends to a very great extent on the capacity of the superintendent to deal with the psychology of the patients. They are often childish, irritable, and complaining, and this is especially well marked in the male patients. A good deal of this can, of course, be put down to the fact that they are now not strenuously employed, and often they are of such a mental and educational standard that they are

unable to fill in the time profitably. Much of this can be avoided by a carefully worked-out time-table; but in spite of the greatest care, outbreaks of grumbling and disciplinary lawlessness arise occasionally.

It is probable also that the mentality is less stable in the consumptive than in the normal individual, as a number develop acute symptoms of delusional insanity while undergoing institutional treatment.



## CHAPTER IX

### INSPECTION

**Position of Patient.**—The examination should take place in a warm, well-lighted, quiet room. The patient may be examined in a reclining position, sitting, or standing. If he is able to bear the strain it is better to have him standing, as the chest is more easily examined in that position. Whether the examination be conducted standing, sitting, or reclining, certain details must be attended to.

The patient should be so placed that the light falls directly on him. His attitude ought to be perfectly easy and natural. The examiner should stand with his back to the light. Some patients have a natural tendency when being examined to stand at "attention"; they inflate the lungs, and keep the chest in a position of forced inspiration, while all the thoracic muscles are in a state of tension. This must be corrected. The patient should stand "easy," in his natural attitude, breathe quietly, and relax his muscles. Unless attention is paid to this much information may be lost, and from what is gained misleading inferences may be drawn. The examiner must himself avoid strained positions, as these will certainly interfere with the delicacy of his percussion and auscultation. The patient's clothing should be removed so as to leave the body bare from the waist upward. When female patients are being examined a soft wrap may, with advantage, be thrown loosely over the shoulders and back, when the front of the chest is under examination, and across the chest while the back is under scrutiny. This lessens the sense of undue exposure which some sensitive patients feel, and does not in any way interfere with the free movement of the chest. The

examination should be conducted leisurely and methodically, each point noted and considered, so that at the end a well-balanced opinion may be expressed as to the condition of the patient. While the patient is undressing, careful attention should be directed to his *general condition and bearing*, as much information can be gained thereby. The manner of undressing may demonstrate an enfeebled condition, such as would indicate serious disease. The whole carriage and gait may be suggestive of a similar condition, or it may be such as to indicate that, if disease is present, it has not yet produced any marked effect on the general physical condition of the patient.

The face should be scrutinised carefully, as it often furnishes valuable information regarding chest diseases. It may appear healthy, plump, and well-coloured, or it may show signs pathognomonic of disease: profound emaciation — malar bones prominent, cheeks and temporal regions hollow, neck long and thin with prominent sternomastoids, eyes sunken in sockets, palpebral margin wide, pearly white sclerotics with dilated pupils like those of a tired child in the evening.

Alterations in colour are noteworthy. The lips may be pale or bluish, and the cheeks may be cyanosed or show capillary injection. These conditions usually indicate some interference with the venous return, such as is caused by emphysema, tubercūlosis, acute or chronic, silicosis, or cardiac disease.

The character of the breathing should also be noted. It may be quiet and normal, or it may be hurried and abnormal either through excitement or disease. It may be shallow, with inspiration interrupted by a quick "catch," such as is found in acute pleuritic conditions. These general points should all be observed before the examination of the chest itself is begun.

### Inspection of Chest.

In inspecting the chest, attention should be directed to:—  
I. The condition of the chest wall. II. The shape. III. The movement.

## I. CONDITION OF THE CHEST WALL

The main points that demand notice in connection with the chest wall are the condition of the skin, the development of the muscular layer, amount of subcutaneous adipose tissue, the presence or absence of superficial veins and of capillary injection.

The skin may be soft, supple, and with a healthy tone about it, or it may be harsh, dry, and tend to pass readily into a cutis anserina condition. These latter states may be indicative of debilitating disease, interfering with the proper cutaneous nutrition, or they may be due to a general unhealthiness, such as is caused by bad environment, uncleanness, or insufficient food. The presence or absence of fine downy or silky hairs in the interscapular area should be noted. These are usually found in weakly, delicate children.

The amount and condition of the subcutaneous fat may be estimated partly by inspection and by palpation. In early disease there may be little diminution, whereas in advanced cases the absence of this adipose tissue causes the skin to lie closely approximated to the bones and to sink deeply into the interspaces. In some cases there is a good layer of subcutaneous fat, but it is flabby and soft, lacking the firmness found in the healthy state. It is a well-known fact that a patient may have a good layer of subcutaneous fat and yet be the subject of extensive tuberculosis.

The muscular layer of the chest is not much altered in appearance in early cases of tuberculosis, but in advanced disease there is atrophy of the pectoral and other chest muscles. If there has been much coughing, the extraordinary muscles of respiration in the neck stand out prominently, and this is often accentuated by the loss of subcutaneous fat. Long-continued coughing also develops the upper part of the rectus abdominis. This is well shown in many cases of emphysema.

The presence or absence of superficial veins coursing over the chest wall should be noted. These are not infrequently seen in the upper part of the chest in cases of tuberculosis. It is commonly assumed that they indicate some obstruction

to the venous return by the intercostal or internal mammary vein. Such obstruction may be caused by the pressure of enlarged intra-thoracic glands. That this is so in some cases is quite correct, but it does not account for all cases, as in many children most of the superficial veins are abnormally prominent. In nursing mothers it is of course quite normal to have enlarged visible veins, due to the temporary activity of the mammary gland.

Capillary injection on the chest wall is fairly common. The injected vessels may be scattered across the front of the chest, but it is much more usual to find them grouped in two situations—first, along the anterior and lateral margins of the thorax, corresponding roughly to the costal attachment of the diaphragm; and secondly, in a small area on the dorsal surface about 4 inches in diameter, which has its centre about the third dorsal vertebra, and has been termed Lombroso's "Varicose zone of alarm." Their causation in the posterior apical region has been attributed to the pressure of thickened pleura on the three upper intercostal veins which are in immediate contact with the pleura. These veins, in addition to draining the intercostal spaces, receive tributaries from the subcutaneous tissue of the back, so that any obstruction to the normal venous return may cause a varicose condition of the superficial tributaries. Their causation along the costal margin is not so easily explained. The significance of these telangiectatic conditions is very difficult to assess. They are seen most frequently in emphysematous or chronic tuberculous conditions, but are at times observed when no abnormal chest conditions can be detected. Their presence may usually be taken as evidence of some interference with the pulmonary circulation, although the alteration in the lung which has produced the condition may give rise to no other physical signs of disease; but as one observes their occurrence at times in the apparently healthy, too much importance must not be attached to them.

## II. SHAPE OF CHEST

The normal chest should be symmetrical on both sides, free from deep hollows in the clavicular region, and with a

smooth, well-rounded contour. A transverse section shows that it is not circular but elliptical, the longer diameter being from side to side. The proportion of the side-to-side diameter as compared with the antero-posterior is roughly about 7 to 5.

Anteriorly the sternum lies in a slight hollow, and is convex from above downward. At the lowest point of the sternum the hollow is more marked, and is known as the infra-sternal depression. The angle formed by the ribs with the sternum should be approximately  $70^{\circ}$  in males, and  $75^{\circ}$  in females. But this may vary somewhat even in what might be classed as normal chests.

The pectoral muscles should be well developed, and when in this state a slight depression is often seen below the clavicle indicating the division between these muscles and the deltoids. Laterally the ribs should sweep round in bold fashion, not too obliquely and with good wide interspaces. The edges of the pectorals and latissimi dorsi should be distinct and firm. Posteriorly the scapulæ should approximate closely to the ribs; and should have the inner margins parallel with each other. They should not have the angles prominent, and these should be at the same level. Perfectly developed chests, however, are rare, and this may be attributed in great part to the absence or insufficiency of suitable physical exercise.

Abnormally shaped chests, although common, are not incompatible with a vigorous life and a healthy state of the lungs. A chest may be abnormal by reason of—(1) congenital defects; (2) imperfect development; (3) disease.

(1) **Congenital Defects.**—Abnormalities due to these defects are not very common. Occasionally one meets a congenital absence of the whole or part of the pectoral muscles on one side. This causes an apparently marked retraction in the affected side, and on a cursory examination might suggest gross underlying disease. The author has seen such a case in a soldier, whose claim for a pension was based on a supposed fibroid tuberculous condition of the right apex, the chief sign of which was the marked retraction of the upper part of the chest. The cause of the retraction was demonstrated by giving the patient some exercises



for the pectoral muscles, and the absence of the muscle on the retracted side was thus made manifest. Occasionally the trapezius muscle is absent or defective, but this is extremely uncommon.

(2) **Imperfect Development.**—It is commonly found that in right-handed persons there is a poorer development of the left side of the chest than of the right, and in persons who in youth have had much clerical work, it is often found that there is a distinct flattening of the left infra-mammary region. This is due to a faulty position having been adopted at work. These points must always be inquired into before assuming that the alteration is due to underlying lung disease.

There are two classical types of chest associated with imperfect development—the flat and the alar chest. These have been looked upon as indicative of a predisposition to, or to the actual development of, tuberculosis. The cause, however, of these chests may more appropriately be attributed to the lack of proper physical exercises. Were a proper system of gymnastics adopted in the schools, and carried on throughout adolescence, few flat or alar chests would be seen.

*The Flat Chest.*—Here the well-rounded convexity of the normal chest is lost; the sternum is flat or may even be slightly concave, the upper ribs sharing in the flatness or concavity. The antero-posterior diameter of the thorax is consequently lessened and the normal expansion during inspiration much diminished.

*The Alar Chest.*—This is often an accompaniment of the flat chest, but not always. Here the striking feature is the prominence of the angles of the scapulæ. These with the vertebral borders project outward from the back unduly. Associated with this, and probably causing it, is an undue obliquity of the ribs which tend more to the perpendicular than normal. The interspaces are narrowed, and the thorax is longer and flatter than usual. A radiogram in these cases will show an undue crowding especially of the upper ribs, one tending to overlap the other. This condition has been graphically described as the “roof-tile” or “waterfall” ribs. In such cases the neck is long and thin, the shoulders sloping, the throat prominent.

(3) **Disease.**—In discussing the changes caused by disease, it appears advisable to introduce the more common alterations met with, as these changes, when present, complicate the diagnosis, although they themselves are not caused by tuberculosis. For the sake of clearness it is well to discuss them under two divisions—Bilateral Changes and Unilateral Changes.

BILATERAL CHANGES are most frequently due to—(1) rickets; (2) Pott's curvature; (3) emphysema.

(1) *Rickets.*—The changes caused by rickets produce the following—(a) the rhachitic chest; (b) the pigeon chest; (c) the chest with Harrison's sulcus; and (d) the chest with the sternal depression.

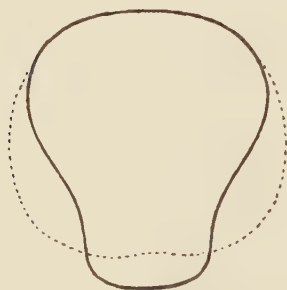


FIG. 17.—Diagram of Rhachitic Chest.



FIG. 18.—Diagram of Pigeon Chest.

(a) *The rhachitic chest* is due to some obstruction to respiration, such as that caused by chronic nasal catarrh, adenoids, or frequent attacks of laryngismus, coupled with the undue softness of the ribs. The obstruction to the free entry of air into the lungs causes a yielding of the ribs at the costal cartilage. The main factor in the yielding is the external atmospheric pressure. As a consequence of this a broad vertical groove is formed at the junction of the ribs with their cartilages. For the production of such a chest a trivial amount of obstruction only is needed, if the softening of the ribs be marked.

(b) The causes which produce the rhachitic are also at work in the production of the *pigeon chest*. It is difficult to explain why in one case a rhachitic chest is produced and at another time a pigeon chest. It is probable that

it depends on the relative amounts present of the softness of the ribs and obstruction to the air entry. In the pigeon chest the rounded curve of the ribs is diminished; they join the sternum at an acute angle, and the sternum itself is pushed prominently forward, so that it projects beyond the general level of the chest and abdomen.

(c) Another extremely common deformity is *Harrison's sulcus*, which consists of a broad depression of varying depth, running transversely across the chest about the level of the lower end of the sternum. It has been stated that the deformity is due also to obstruction to the inspiration in rickety children. The author is of opinion that the deformity is caused by other factors altogether. It appears to be chiefly due to the rickety child sitting, as it often does for a prolonged period, in a bent-forward position; the abdomen, usually swollen and prominent, presses upon the lower margin of the ribs, and actually tilts them forward. The upper part of the thorax, strengthened by the sternum, is pressing downward and inward, and the lower costal margins being pressed outward, the result is a bending of the thoracic wall where these two forces meet. Let the explanation be what it may, the deformity is very common, and traces usually remain in adult life.



FIG. 20.—Diagram showing Sternal Depression.

(d) *In the sternal depression* the lower portion of the sternum lies at the bottom of a conical-shaped depression, the cartilages of the lower ribs sloping inward and backward from the end of the ribs to join the depressed sternum. The usual explanation of obstruction to breathing in a rachitic child is again advanced as the cause of this deformity.

(2) *Pott's Curvature*.—In cases where there is pronounced angular curvature of the spine in the dorsal region, the chest takes a characteristic shape. The upper ribs are crowded upon each other, the sternum pushed forward, and the antero-posterior diameter of the thorax much increased. In such

chest's movement is much limited, and there is also often considerable displacement of the thoracic organs, and interference with their functional activity. A simple attack of bronchitis in such a chest is apt to be a prolonged illness, and moist crepitations are heard for weeks at the bases of the lungs. This is a point to be remembered in dealing with bronchitis in hunchbacks as, because of this, a wrong diagnosis of tuberculosis is often given.

(3) *Emphysema*.—In emphysematous conditions of the lung the thorax as a rule becomes considerably enlarged and is fixed in a position of extreme inspiration. The shoulders are raised, the ribs more horizontal, with much widening of the interspaces. The substernal angle is much increased, and the chest as a whole more circular in shape and shorter than normal.

UNILATERAL CHANGES.—(1) *Enlargement*.—One-sided enlargement of the chest is found (*a*) in cases of pleurisy where there is a large effusion of fluid into the pleural cavity; (*b*) in some cases of tumour of the lung; (*c*) in cases of pneumothorax or hydro-pneumothorax; (*d*) in scoliosis.

On the enlarged side the ribs are more horizontal than usual, and the interspaces much wider. There may be some amount of cardiac displacement. If the effusion or pneumothorax is on the right side, then the heart is displaced to the left; and if the effusion is in the left side, then the heart is displaced to the right. The amount of cardiac displacement depends to a great extent upon the amount of effusion or of air in the pleural cavity.

(2) *Diminution or Retraction*.—Unilateral shrinking of the thorax is found (*a*) in cases of old-standing fibroid tuberculosis of the lung; (*b*) in cases of pleurisy with effusion, where the fluid has not undergone absorption but has become organised; (*c*) in chronic interstitial pneumonia; (*d*) in the concave side of a scoliosis; (*e*) in old cases of empyema; (*f*) in collapse of the lung after penetrating wound. The explanation of the shrinking of the chest is that the new-formed tissue in the chronic tuberculous condition or in organised pleural effusion undergoes contraction—as all cicatricial tissue does—and, in contracting, pulls in the adjacent ribs. There is also some



FIG. 22.—Emphysematous Chest.





considerable displacement of the heart, but the displacement in these cases is toward the shrunken side.

In tuberculous disease of the lung in the earlier stages there may be no alteration in the shape of the thorax. As the disease advances, retraction above or below the clavicle may be noted. In some cases it is extremely slight, but in others it is pronounced, the clavicle standing out prominently with hollowing of the parietes above and below. It is a mistake, however, to conclude that every case of retraction in the apex is due to tuberculosis of the lungs. When found under the clavicle, it may be due to insufficient development of the pectoral muscle or to scoliosis. In all cases of slight flattening the presence or absence of lateral curvature should be carefully determined. A considerable unilateral flattening is often seen in cases of hemiplegia or infantile paralysis. With these exceptions, marked unilateral apical retraction can as a rule be safely attributed to the cicatricial contraction of tuberculosis of the underlying lung. When the retraction is bilateral and equal in both apices it is difficult, on inspection alone, to decide whether it is due to underlying tuberculous cicatrization, to faulty development, or to carriage. It may also be seen in elderly patients who are the subjects of emphysema owing to elevation of the clavicles.

As a rule, in tuberculosis these local changes are first observed anteriorly, but as the disease advances, well-marked flattening may be observed in the supra-spinous area on the affected side. This flattening is due to atrophy of the supra-spinous muscle, and always indicates a lesion of some considerable age. The writer has often tried to determine the age of the lesion which will produce supra-clavicular retraction, and has concluded that, when the retraction is slight or moderate in degree, at least eight months have passed since the beginning of the pathological process in the apex. When the retraction is pronounced the underlying lesion may have been of several years' duration.

When the disease is extensive, and fibrosis predominant, marked retraction may be observed in the infra-scapular area, causing a shrinking of the lower part of the chest, with a pronounced furrow at the inner border of the scapula, or a

marked projection and lowering of the angle of that bone. Such retractions are chiefly found in fibroid phthisis, or long after a pleural effusion which has been incompletely absorbed. When the disease is widespread throughout the upper lobe, well-marked retraction or hollowing may be found in the second, third, and fourth interspaces. Should the left lung be affected, then, in addition to the retraction, there may be seen pulsation in the retracted interspaces. This is due to the shrinking lung exposing the heart's surface to the chest wall, and the impulse is communicated directly to it without the intervention of the spongy lung.

In some cases, when the patient is being examined a drooping of one shoulder is the first point which attracts attention. This drooping may be due to a variety of causes. It may arise from extensive fibrosis of the lung, causing a shrinking of the side, with narrowing of the ribs, or it may be, and often is, due to scoliosis. On the other hand, it may be due to partial paralysis of the trapezius, the result of injury to the spinal accessory nerve in an operation for cervical adenitis. When due to pulmonary disease it is always an indication that the changes in the lung are old and extensive.

Inspection may also reveal some undue local prominence of the chest wall, but the conditions causing this are rarely due to tuberculosis, and may be dismissed in a word. Emphysema often causes some fulness in the supra-clavicular regions. In cardiac hypertrophy there may be some prominence of the precordium; aneurism of the arch of the aorta may produce some bulging above or on either side of the manubrium, according to the situation and size of the aneurism. These swellings may be formed, of course, in other parts of the chest wall, but the expansive character of pulsation always reveals their nature. Localised swellings may also be due to abscesses, bone tumours, lipomata, etc., but the diagnosis of these is beyond the scope of this work. As an instance of rare localised swelling, surgical emphysema may be seen following a paracentesis thoracis for pleural fluid or after artificial pneumothorax.



FIG. 23.—Drooping of Shoulder, due to injury to spinal accessory nerve in an operation for cervical glands.



FIG. 24.—Drooping of Shoulder in a case of scoliosis.





### III. MOVEMENT

In addition to the general condition and shape of the chest, attention must be paid to its movements. In health both sides move equally, but when the lung is affected with tuberculosis this equality is disturbed. In early disease it is often noted that, on the affected side, the movement lags behind that of the other, or that the excursion of the chest wall is limited. This lagging may be due to lack of tone in the muscles overlying the seat of disease or to actual cicatrization of the lung. It may only be noted at the beginning of inspiration, and the ultimate excursion of the affected side may be quite equal to that of the other. In advanced tuberculosis the chest wall on the diseased side may be quite immobile.

When the base is involved there is limitation of the movement in that region of the chest wall which again varies from a slight degree to practical immobility.

In some patients the movement in the upper part of both sides of the chest is extremely limited. This may be due to underlying disease in both lungs, or to the fact that the patient has never practised deep breathing.

For noting fine shades in the difference of movement on the two sides, the observer ought to stand behind the patient, who should be placed in a good light, with the head slightly bent forward. The examiner should then direct his gaze so as to simultaneously compare the movement on both sides. A little practice is necessary for this, but when the art is mastered very minute differences can readily be detected. Better results are obtained in this way than by attempting to estimate the movement when the examiner stands in front of the patient.

The type of respiration in emphysema is noteworthy. In this disease the chest is in a position of inspiration, that is the chest wall is expanded to nearly its full extent, and the diaphragm is depressed considerably more than in health. As a consequence of this, when the patient attempts to inspire, the chest cannot readily expand further, but the extraordinary muscles of respiration (the sterno-mastoids, the scaleni, etc.) are called into play, and the chest as a whole

is pulled upward. At the same time the diaphragm descends a little, and so the thorax is enlarged from above downwards. There is much effort, but not a corresponding increase in the respiratory capacity. Inspiration in emphysema is characteristically short, while expiration is unduly prolonged. This type of respiration, so common and so easily recognised, is emphasised in order to impress the student, for emphysema masks the tuberculous changes, and such a chest demands extra care during examination in order that underlying tuberculous disease be not overlooked.

## CHAPTER X

### PALPATION

THIS part of the examination of the patient should never be neglected, as much can be learned from it. A very slight elevation of temperature can be detected by an experienced examiner the moment he places his hand on the chest wall. An opinion can be formed as to the condition of the subcutaneous fatty layer, and of the muscular covering of the thorax. Is the adipose tissue thick or thin, firm or flabby? Are the muscles well-developed and in good condition, or are they ill-developed and poor? These questions are settled by palpation. In cases where the ribs still preserve some considerable flexibility, it is often possible to detect an increase of resistance where there is an underlying solid lung. This is a point of interest, but of course the results of palpation in such a case cannot be compared, for accuracy, with percussion over such a lung. Palpation will reveal the presence or absence of cardiac thrills, and, in any localised swelling, the pulsation or thrill which might indicate the presence of an aneurism.

By palpation also we detect the character of vocal or tactile fremitus. This is best felt in males, and particularly in those with a deep voice and a thin chest wall. In women and children it is often so faint that it cannot be detected. It is best elicited by getting the patient to repeat slowly and in a low tone of voice such words as ninety-nine, or one, one, one.

The observer's whole hand should be placed on one side of the chest, and then a comparison made with the other side, using the same hand. When localised areas are under examination, the finger-tips only should be used and applied lightly. The strength of the vibrations is much affected by

the amount of adipose tissue present in the chest wall. A thick layer may well nigh obliterate it altogether. The vocal fremitus is not felt in all parts with the same distinctness; it is best felt in the upper anterior and lateral aspects of the chest. It is less marked in the cardiac region, the lower lateral areas, the bases, the apices posteriorly, and is most diminished in the scapular areas. The normal difference which exists between the apices should be borne in mind. It will be remembered that the fremitus is much more marked in the right apex than in the left, both anteriorly and posteriorly, and more marked in the right interscapular area than the left.

In early tuberculosis it is often impossible to decide whether the increase of the vocal fremitus on the right side is physiological or pathological, whether, in short, the underlying lung is healthy or diseased. This point can only be decided by carefully weighing the evidence presented by other methods of examination. A definite increase on the left side is often of great assistance, especially if the fremitus is actually greater on the left than on the right side.

The vocal fremitus can undergo three changes: it may be increased, it may be diminished, or it may be absent, and, according as one or other of these changes is found, we draw certain deductions. It is increased, speaking generally, when there is some underlying solidification of the lung, as in pneumonia or tuberculosis, or where there is a superficial cavity. It is also increased in emphysema. Occasionally it will be found that in some solid conditions of the lung the fremitus is diminished. It is diminished or absent (*a*) when the main bronchus is occluded by pressure of an aneurism or tumour, or where there is stenosis of the bronchus; (*b*) in pneumothorax; (*c*) in pleural thickening; (*d*) in some cases of acute pneumonia; and (*e*) in pleural effusion.

Certain sounds produced in the thorax communicate a vibration to the chest wall, and can be felt there. These are (*a*) loud coarse rhonchi, (*b*) pleural friction, and occasionally (*c*) cavernous râles.

In some rare cases of pleural effusion, empyema, and hydatid cyst, distinct fluctuation can be felt.

The cervical glands should always be examined, and especially those lying in the supra-clavicular area, as sometimes in apical tuberculosis these are enlarged and can easily be detected lying behind the clavicle. The reason for the glands being affected is that they drain the parietal pleura, and should it become tuberculous or adhesions be set up between the visceral and parietal layers, as in a case of tuberculosis of the apex, then the deep glands behind the clavicle may show evidence of disease also.

The condition of the muscles demand some attention when palpating the patient. It is a well-known fact that in inflammatory conditions of the abdominal organs the overlying muscles are in a state of spasm, more or less marked. It would appear that an inflammatory condition of the lung produces the same effect upon the chest muscles. This can be noted in cases of pleurisy, when the side affected moves less than the other, and X-ray examination will show the diaphragm on the affected side to be greatly limited in movement as compared with the healthy side. Although the inflammatory process in tuberculosis may be less pronounced than in peritonitis or pleurisy, yet it is active enough in many cases to produce muscle spasm. This can be felt by placing the tips of the fingers over the muscle and gently rolling the skin over it. They feel thicker and firmer than those of the other side. This muscle spasm is a sign of some slight aid in diagnosis, and its value is increased when the possibility of physiological increase of the muscle owing to the patient's employment can be excluded. The author has not infrequently demonstrated its value to students by asking one of his assistants to diagnose the affected side by palpation only. To the uninitiated this looks impressive, but muscle spasm is only a very minor auxiliary sign.

In early tuberculosis the condition when present is best detected in the trapezius, but it can also be elicited in the pectorals and the sterno-mastoids. As the disease passes into the advanced stage, the spasm disappears; the muscle loses tone and undergoes some degeneration atrophy. This is readily noted on palpation—the muscle



is thinner and softer, and this contributes to the hollowing seen in the apical region in advanced disease.

When palpating, it is well to make a habit of noting the condition of the walls of the blood-vessels. They may show atheromatous changes, that may have an important bearing in a case of hæmoptysis.

## CHAPTER XI

### PERCUSSION

PERCUSSION is "the art of striking a part of the body so as to beget a sound useful for the discovery of disease." To become an expert in it requires much prolonged practice. A delicacy in manipulation is needed, and an ear trained to detect the slightest alteration in the sounds produced. Percussion depends on the fact that when a body is tapped certain vibrations are set up in it, and these are communicated through the air to the ear. The sound which is elicited depends on the force which is used and the structure of the body percussed. In the thorax the note produced depends, in part, for its characteristics on whether the underlying structures are solid like the heart, the liver, etc., or whether they contain a large amount of air like the lungs. In percussing over the lungs themselves the note also varies according as to whether the lung is normal, or solid as in pneumonia, or solidified in small patches as in some cases of tuberculosis, and it varies also according to the thickness of the chest wall.

Before beginning to percuss, it is essential, as was noted in inspection, to see that both the patient and the examiner are in an easy, comfortable position. It is customary to discard all percussing instruments, and use only the fingers. The middle or index finger of the left hand is usually used as the pleximeter, and it should be pressed firmly against the part of the chest which is being percussed. It is essential that this should be so, for if the finger be not firmly applied no reliance can be placed on the percussion. When comparing different sides the same amount of pressure must be used, else the result will not be trustworthy. The other fingers of the left hand should not rest on the chest, as

they may interfere with the vibrations. The stroke should be delivered on the second phalanx, or for very fine percussion on the first, behind the root of the nail. The middle finger of the right hand should be used as the plessor, and the finger should be bent so that the stroke is delivered with the tip and not the ball of the finger. The direction of the stroke should be perpendicular to the pleximeter finger, and it should be delivered from the wrist and partly by the percussing finger itself. A light stroke is always preferable to a heavy one, unless in the supra-scapular area where, on account of muscular development, a heavier stroke is necessary to elicitate the pulmonary note. In superficial dullness, or where the dullness is of small extent, a heavy stroke would set up vibrations in the surrounding and underlying lung such as might altogether hide the note from the dull area, which would consequently be missed. In percussing the liver dullness, it is well known that light percussion will give a resonant note down to the level of the sixth rib in the nipple line, whereas with heavy percussion the note begins to be affected as high as the fourth.

Strict attention must be paid to these details, otherwise the comparison of the notes will be faulty. There must be the same pressure of the finger on the chest wall, the same force and direction of the blow, and the same part of the pleximeter finger must be struck. It should be recognised that there is no exact standard percussion note which can be used for comparison in different individuals. Each patient must be treated as a separate entity. The normal percussion note is best heard in the second or third interspace anteriorly or in the upper part of the axillary region, and the student would do well to practise on all the normal chests he has access to, so as to familiarise his ear with the note.

In order to appreciate slight variations of the sound, it is well to form the habit of analysing it. Percussion tones have four characteristics: (1) intensity or loudness; (2) pitch; (3) quality; (4) duration.

(1) *Intensity* depends on the force of the blow and the condition and volume of the vibrating material underneath. Therefore, in comparing two corresponding areas, if the same force be employed in percussing, and the resulting



FIG. 26.—Position of hands when percussing apices from the front.



FIG. 27.—Position of patient for percussion of chest posteriorly.



FIG. 28.—Position of hands when percussing the apices from behind.





note is not so intense upon one side, the natural inference is that there is some alteration in the underlying structure. (2) *Pitch* refers to the length of vibrations producing the note. Long vibrations produce a low tone, shorter vibrations a higher tone. Intensity has no relation to pitch, as a weak note may have the same pitch as a strong one. A healthy pulmonary note is comparatively low, and in pathological conditions, such as tuberculosis, the pitch as a rule rises, the rise depending on the amount of solidification present. (3) *Quality*. It is as difficult to describe the quality of the normal percussion note as it is to describe that of a violin. It has to be heard to be appreciated. In the chest the percussion sound is spoken of as the pulmonary note. (4) *Duration*. This feature is of so little practical importance that it may be ignored altogether. Attention to the properties of the percussion tones and a habit of analysing them will enable the observer more readily to note any variation in them, and to be more confident in the conclusions he draws.

### Extent of Pulmonary Resonance.

It is important that the normal boundaries of the pulmonary resonance should be familiar to the examiner, otherwise pathological conditions involving the margin of the lungs may be easily overlooked. In delineating the boundaries the pleximeter finger should be kept parallel to the edge of the lung. The normal resonance of the apex should be approximately as follows:—Posteriorly, it begins about the level of the seventh cervical vertebra, then passes upward and outward until it crosses the trapezius at a point from  $1\frac{1}{2}$  to 2 inches above the clavicle, then it passes downward and inward to a point behind the middle of the manubrium sterni. It is difficult, as a rule, and at times impossible to get the exact limit in front, as the tracheal resonance complicates matters considerably. In practice the anterior boundaries are rarely defined.

*Krönig's area of resonance* should always be mapped out. This is the area of resonance above the apex, and to find its limits the lung is percussed from the central area of

resonance outwards, and a line drawn to mark the division where the resonant area is left. This gives us the outer boundary. In the same way, by percussing inward the inner border is defined and a line again drawn. This area can also be defined with great exactness by "transmitted percussion." If one observer places his stethoscope about the middle of the second intercostal space in front, while another percusses softly over the apex, gradually moving outward or inward to the limit of the resonant area, the alteration in the note can readily be distinguished by the observer auscultating in front. The measurement in health at the narrowest part is usually stated as being 5 cm.

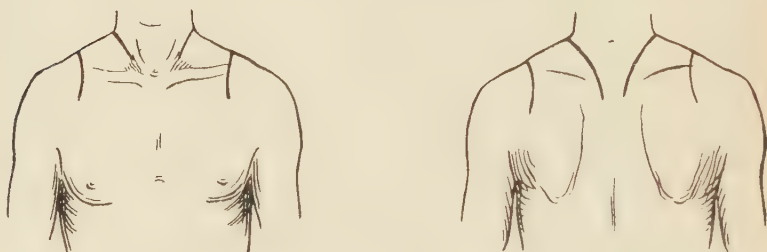


FIG. 29.—Krönig's Area, anteriorly and posteriorly.

If under 4 cm. the apex is not normal. This area is of importance. It does not represent the actual apex of the lung, but it does represent the projection of vibrations from the underlying resonant area on to the skin surface. In tuberculosis it is usually diminished on the affected side, and although the resonant area does not coincide with the actual apex, yet this does not interfere with the diagnostic value of any diminution which may be found, as the area is quite comparable on the two sides.

Another point to be noted here is the difference which is shown on the area of resonance between inspiration and expiration. The outer boundary toward the acromion remains practically unchanged, but the inner boundary moves considerably. The limits of the area are marked during ordinary quiet inspiration, and then the patient asked to take a deep breath and to retain it. While he does this

the inner margin is again rapidly percussed and the new line showing the increase of the resonant area mapped out. Much practice is required to do this with any degree of exactitude, and as a rule it is only performed by those practitioners who specialise in chest work. In tuberculosis of the apex, the *tidal movement*, as it has been termed, that is the difference between inspiration and expiration, as shown in Krönig's area, may be much diminished or altogether obliterated.

The anterior borders of the lungs meet behind the manubrium sterni and continue behind that line until the

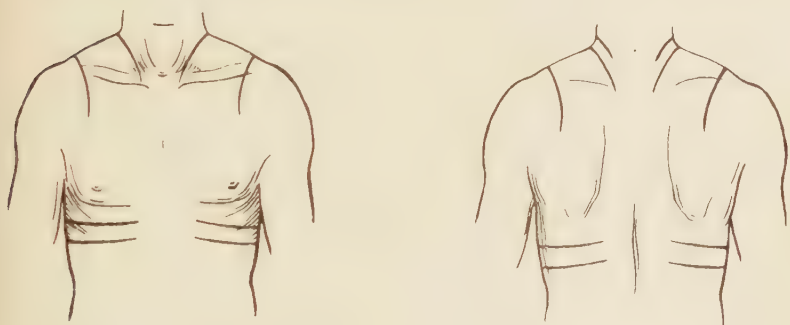


FIG. 30.—Tidal Movement at apices and bases.

fourth costal cartilage is reached. On the left side the margin of the lung runs outward and downward, leaving the cardiac surface uncovered. This area forms the superficial cardiac dullness, and it is detected best by light percussion. In shape it is roughly triangular—the right border corresponding with the mid- or para-sternal line; the base is an extension of the line that forms the upper boundary of the liver; and the third side is a line drawn from the junction of the fourth costal cartilage and the sternum to meet the base line at a point corresponding with the apex beat. On the right side the anterior margin of the lung is continued downward to about the level of the fifth rib, then it slopes outward and downward across the sixth interspace and into the lower axillary region.

In the nipple line the lower limit is the sixth rib on quiet

respiration. Then it passes outward and downward until in the axillary line it reaches the eighth. Posteriorly the limit is about the eleventh rib on the left side and the tenth on the right. In the diagnosis of tuberculosis exactness in every detail of the examination is essential, so the student should practise carefully, and as frequently as possible, the delineation of the normal boundaries. It should be noted that the lungs, more especially in the lateral and posterior regions, do not fill the whole of the pleural sac on ordinary respiration, but that there is a considerable space, approximately 3 inches in the axillary region, where the lungs can descend into the pleural sacs on deep inspiration. The limits of the lungs can be outlined posteriorly so as to show the difference between inspiration and expiration in this region also—a fact first noted by Krönig. This difference is of considerable assistance, as will be seen later, in estimating the amount of retraction or limitation of movement, which may be caused by various forms of pulmonary disease.

**Regional Differences in the Normal Percussion Note.—**

It should be recognised that the percussion note is not identical in the various regions of the chest. A recognition of the normal differences will greatly assist the student when he comes to compare them under diseased conditions, and will also impress on him the necessity for comparing corresponding areas of the chest. The supra-clavicular must be compared with supra-clavicular, and infra-scapular with infra-scapular, and so on. The percussion note is of greatest intensity in the infra-clavicular area, extending from the clavicle to the third rib. The pitch is low and the quality is a pure pulmonary one. Both sides are the same. Occasionally a slight tympanitic quality can be detected toward the upper and inner part. This is due to the proximity of the bronchi. In children, a distinct dullness is often noted at the inner margin of the first and second interspaces on the left side. This is sometimes due to prominence of the great vessels, and, at other times, to bronchial glands. This condition is often seen in delicate children, where there may be a flat chest and a deficiency in the pulmonary expansion. When this dullness is met with, it must not be at once assumed that it is due to underlying tuberculous disease.

In the *supra-clavicular area* the note is less intense and the pitch slightly higher. At the inner part, owing to the proximity of the trachea, it becomes slightly tympanitic. The main characteristic of the supra-clavicular note is the loss of intensity. This is due to the small area of resonating lung underneath. The alteration of intensity and pitch varies in different individuals, just as the apex of the lung varies in the distance it projects above the clavicle. In some it is  $\frac{1}{2}$  inch, and in others  $1\frac{1}{2}$  inches. Now the supra-clavicular area is of great importance in early tuberculosis; hence it is essential, in forming a correct opinion on any given patient, to remember the normal alteration in the percussion note. It is extremely difficult, and indeed at times impossible, to state whether a given percussion note in the supra-clavicular areas is normal or abnormal. In the *clavicular area* the note in the centre of the clavicle is slightly less intense than in the infra-clavicular area. Toward the acromial end the intensity diminishes very much, while toward the sternal end the pitch rises, and a tympanitic quality is added to the note.

In the *mammary region* the differences between the two sides are so distinct that they may, with advantage, be treated separately. On the left side the heart alters the note to a very great extent. The limits of the superficial cardiac dullness have already been given, and here the note is quite dull. In addition to the dull area there is an area with altered percussion note around the heart. This is where the edge of the lung overlaps the organ; and while the lung is of sufficient thickness to give a resonant note to light percussion, yet heavy percussion shows a distinct alteration. This is the deep cardiac dullness. Its limits are approximately as follows:—On the right side, the outer edge of the sternum; on the left side the alteration is noted as high as the third rib, or in children the second interspace; from this it runs outward and downward to a point about  $\frac{1}{2}$  inch to the left of the apex beat. The base line is formed by a continuation of the line of the liver dullness. On the right side the percussion note is at times interfered with by the development of the pectoral muscle, so that its intensity, compared with the other side, is slightly diminished. With light percussion a resonant note can be detected to the level of the sixth rib in the nipple



line, where it passes in to the liver dullness. On heavy percussion the alteration can be detected as high as the fifth or even the fourth rib—the intensity being lessened and the pitch heightened.

In the *sternal area* the note is resonant, and for all practical purposes is unchanged from the second to the sixth rib—the sternum being an excellent conductor of sound. In the *lateral region* the note is nearly identical with that obtained in the infra-clavicular area. In the *infra-scapular area* the note, in thin persons, approximates to that obtained in the axillary region, but in stout persons the intensity is very much lessened. Indeed, in excessively fat individuals it is almost impossible to get anything approaching a pulmonary note. In the *scapular and inter-scapular areas* the note is relatively dull, the intensity being diminished, and the pitch slightly raised. In the *supra-spinous areas* in muscular patients the note is often very much lessened, and heavy percussion is required to elicit it. Even in thin persons the note in this area is distinctly less resonant and higher in pitch than the note in the infra-clavicular region.

In comparing the two sides it should be noted that there is a minute difference in the apices. The right is higher in pitch and less intense. It has to be admitted that much delicacy in touch and keenness in the perception of minute differences in sound are necessary to note these changes, and in very many cases there is no noticeable alteration. Indeed, it would almost appear to be safer to assume that whenever a distinct alteration between the notes is detected in the apices that the alteration points to the presence of some disease rather than to its being a physiological difference.

#### Changes in Disease.

The percussion note may be altered in various ways by disease. Its volume may be increased or diminished, and its quality may be changed. The volume is increased in emphysema, in pneumothorax, and at times in cases of pleural effusion, above the fluid level. Here a slight tympanitic quality is sometimes added, and this has been



termed Skodaic resonance. It is often increased, also, when a cavity is near the surface of the lung—indeed, at times a cavity can be detected by the tympanitic quality of the percussion note. The volume of the sound is diminished in cases of effusion into the pleural sac, either serous or purulent; in cases of thickened pleura; or when there is some consolidation of the lung, as in pneumonia, tumour, tuberculosis, infarction, or atelectasis. The alteration of the note varies from the absolutely dull note of an extensive pleuritic effusion to the hardly perceptible alterations which may be found in early tuberculosis.

An important alteration in the quality of the percussion note is that known as tympanitic resonance. This is found at times over superficial cavities, and closely resembles the "stomach note." To produce this it is usually assumed that the cavity must be at least 2 ins. in diameter, with smooth lining walls. In percussing a cavity, sometimes the pitch of the note alters on the patient opening or shutting the mouth. In such cases the pitch rises when the mouth is open (Wintrich's sign). At other times the note alters with a change of position (Gerhardt's sign). The note rises as the patient passes from the recumbent to the erect position. This is supposed to be due to the presence of fluid in the cavity. These changes in the pitch noted over cavities are in practice rarely observed, and more reliance is placed upon auscultation for the diagnosis of a cavity than upon percussion. Another alteration in quality is the "cracked pot sound," which has been likened to that obtained by clasping the hands loosely together and striking them over the knee, thereby expelling some of the air enclosed by the hands. It is found occasionally in percussion over superficial cavities. It may also be detected in the chest of a healthy infant, and in the lung above the level of pleural effusion. The sound is supposed to be due to the sudden expulsion of air through the glottis. The importance of the cracked pot sound need not be emphasised, but it should be noted when it is present.

## CHAPTER XII

### AUSCULTATION

AUSCULTATION may be practised by applying the ear directly to the chest wall with a soft towel intervening, or by using some form of stethoscope. Thus we have direct or immediate auscultation, and indirect or mediate. The direct method is rarely used now, but it is advisable that the student should familiarise himself with it. In the indirect method, a single or a binaural stethoscope is used. The stethoscope surpasses the direct method for ease and convenience, and the binaural for the same reasons surpasses the single stethoscope. In the single wooden stethoscope the sound vibrations are carried from the chest to the ear partly by the central column of air and partly through the wood of the instrument itself. In the binaural, however, the sound is conveyed solely by the column of air.

In choosing a binaural stethoscope the student should see that the curved end suits his auditory meatus; that the ivory ear pieces are of sufficient size to prevent them slipping too far into the ear, and that the openings in them are of some considerable size. The rubber tubing should be thick enough not to kink easily, and should be of sufficient length—about 10 ins.—to enable the examiner to examine both sides of the chest without changing his position. All the parts of the stethoscope should fit accurately and firmly together.

The illustration shown is one commonly employed, but use and wont have a great deal to do with the particular instrument preferred. The bell of the stethoscope should be applied firmly and evenly to the chest wall, care being taken that every part of it is in contact with the skin surface. If the chest is covered, even to a slight extent, with hair, or if

the skin be dry and scaly, in order to avoid local friction sounds, which simulate intrapulmonary crepitations, the part should be smeared with vaseline. The room in which the examination by auscultation is conducted should be quiet, as it is extremely annoying and disturbing to attempt to auscultate a chest with the noise of continual passing traffic. Not only is it disturbing, but it may altogether prevent fine work from being done. An essential point in a tuberculosis dispensary is that it should be situated in a quiet street and not in a noisy thoroughfare.

When auscultating, the examiner should concentrate attention on the sounds he hears, and the patient should be directed to breathe quietly and naturally. All sources of adventitious sounds, such as those produced by the rubbing of the clothes near the part auscultated, must be avoided, and care should also be taken that the sound caused by swallowing saliva or sputum is not mistaken for crepitations. By auscultation we determine certain facts: (1) the quality and variety of the respiratory sound; (2) the character of the vocal resonance; (3) the presence or absence of any accompaniments or adventitious sounds and their nature and quality. We shall now examine each of these more precisely.

**The Respiratory Sound.**—Over the healthy lung the sound is of a soft, rustling character—a sound peculiar to



FIG. 31.—Common form of Stethoscope.

itself, and is known as vesicular breathing. In listening over the trachea, and often over a solid lung, the breathing is of a very different character from the vesicular variety. It loses its fine rustling quality, and becomes hollow like the whispered "ch" in loch. This variety is termed bronchial breathing. These two varieties are quite distinct from each other, and cannot be confused, but there are types of breathing which are neither vesicular nor bronchial. They have some of the qualities of each. Hence they have been termed indeterminate, metamorphosing, or broncho-vesicular. Thus we have three forms of the respiratory murmur: (a) vesicular breathing; (b) bronchial breathing; (c) broncho-vesicular breathing.

In listening to any respiratory murmur, a careful comparison is made of inspiration and expiration, and attention is given to the intensity, pitch, quality, and duration of the sound. With these points before us we can now further examine each type.

(a) **VESICULAR BREATHING.**—There is no absolute normal standard—it varies slightly with the individual. It is heard best in the axillary and infra-scapular areas. It varies with age, with the thickness of the soft tissues, and with the force and rapidity of respiration. There is, however, always a definite relationship between the length of inspiration and of expiration. During inspiration the pitch is low and the duration is of the same length as the inspiratory effort. Expiration follows inspiration without any distinct pause in ordinary quiet breathing. The intensity is less; the pitch lower; the quality is less rustling or less vesicular, and the duration is about  $\frac{1}{4}$  or  $\frac{1}{3}$  less than inspiration. In general it may be stated that the intensity of the vesicular murmur is in proportion to the rapidity and depth of the respiratory effort. The quicker the breathing the greater the intensity, and the expiratory phase becomes proportionately lengthened.

There are a few varieties of vesicular breathing, of which the following are noteworthy:—

(1) *Puerile Breathing.*—The chief alteration here is that the intensity is increased. There is also a slight change in the relative durations of the inspiratory and expiratory murmurs, —expiration being slightly prolonged. Puerile breathing is

probably due to the thinness of the chest wall, and to the increased frequency in inspiration which is found in children. It is heard normally up to puberty.

(2) *Harsh Breathing*.—This is practically identical with the puerile form, but is found in adults. Here the inspiration is increased in intensity, and the pitch higher than the normal, and the expiration prolonged. It is said that this type of breathing indicates some loss of elasticity in the lung tissue. It is also found when there is some slight consolidation in the lung.

(3) *Cogwheel or Interrupted Breathing*.—In this variety there are one or two distinct breaks or jerks in the continuity of the inspiratory murmur. This is said to be due in some cases to loss of elasticity of lung tissue caused by such a disease as tuberculosis, or it may be due to nervousness, but this does not explain all cases. Careful observation will show that these breaks are always synchronous with the apex-beat, so that it appears more likely that they are due to the heart systole affecting directly either a large or small bronchus. The significance of cogwheel breathing is of little importance. Its presence should be noted merely as a fact.

(4) *Feeble Breathing*.—In some patients the respiratory murmur is very indistinct, more particularly in the posterior part of the chest. This may be due to the thickness of the parietes or to insufficient chest expansion. Its presence as a sign of disease will be discussed later.

All regions of the chest do not exhibit the same vesicular breathing. The most important difference is to be found in the supra-clavicular areas, and it is just in this region where the earliest signs of tuberculosis have to be searched for, so that it is of importance that the student should be fully cognisant of the normal differences. If the axillary region be taken as the standard, then the chief variations in the supra-clavicular are as follows:—

*In Inspiration.*

Intensity is lessened ;  
Pitch is slightly higher ;  
Quality slightly less vesicular ; and  
Duration shortened.

*In Expiration.*

Intensity is slightly increased ;  
Pitch is still slightly higher ;  
Quality more bronchial ; and  
Duration prolonged.



In addition to these changes, it must be carefully noted that there is a marked difference between the respiratory murmur of the two apices. In the right, the intensity is much increased and the breathing becomes more of a bronchial type, and this constitutes one of the most difficult problems that the practitioner meets with in the diagnosis of tuberculosis. If a patient has a respiratory murmur in the right apex approximating the bronchial type, is this physiological or pathological? The writer believes that an almost pure bronchial breathing is quite compatible with a healthy lung. Much experience is necessary, and much time must be spent on other signs and symptoms, and other methods of examining the apex adopted, before a definite opinion can be given as to the actual significance of bronchial breathing in the right apex. To classify a patient as suffering from tuberculosis simply because an approximation to bronchial breathing is heard in the right apex, is wrong, and shows an ignorance of the extent of physiological variations in the respiratory murmur. The cause of this variation was formerly supposed to be due to the difference in shape and position of the right eparterial bronchus, but it now seems conclusively proved that it is due to the proximity of the right apex to the trachea. In the left, the apex is separated from the trachea by the left innominate and supra-clavicular arteries, as well as by a comparatively thick layer of connective tissue. On the right side, however, the apex is only separated from the trachea by a thin layer of connective tissue, hence sounds generated in the larynx are communicated more readily to the right apex.

In the scapular area the respiratory murmur is less intense than in any other area, so that slight alterations here are not so easily noted. In the inter-scapular areas the intensity is good, although not quite so well marked as in the axillary or infra-clavicular areas. There is also a slight difference between the two sides, but not so marked as in the supra-clavicular areas.

(b) BRONCHIAL BREATHING.—This is heard best over the trachea or bronchi, in thin persons or children, and over the vertebral column from the 7th cervical to the 3rd or 4th dorsal. The characteristics of bronchial breathing are

as follows:—The fine rustling sound of vesicular breathing is lost, and is replaced by a hollow tone, as already stated, like the whispered “ch” in loch. There is a well-defined pause between inspiration and expiration. The expiratory sound is more intense than that of inspiration, is slightly higher in pitch, and is longer in duration. It should be observed that mere loudness is not an essential characteristic—the hollow quality is the important point.

The varieties of bronchial breathing are three in number, and depend on difference of the pitch. They are:—

(1) *Low-pitched or Cavernous Breathing*.—This is heard over fairly large-sized cavities. Amphoric breathing is practically identical with cavernous breathing, except that there is a musical quality added. It is found where there is a large cavity, with smooth walls, communicating with a bronchus. (2) *High pitched or tubular breathing* is heard in its perfection over the solid area of lung in pneumonia. (3) *Medium pitch* has no special significance.

(c) BRONCHO-VESICULAR BREATHING. — This, as its name suggests, is a combination of the vesicular and bronchial sounds. It is found often in commencing consolidation in tuberculosis, when a layer of healthy lung intervenes between the solid portion and the chest wall, or where the patch of consolidation is small. As a rule, the inspiratory sound is vesicular, while the expiratory is bronchial in character, but occasionally the inspiratory sound alone partakes of the bronchial character.

**Vocal Resonance.**—Under this heading we take up the study of the voice sounds by the stethoscope. When a patient pronounces such words as “ninety-nine,” auscultation over pulmonary tissue, in the infra-scapular area for example, reveals a somewhat muffled buzzing sound, and if contrasted with that heard over the trachea it will at once be noted that the sound has lost in clearness and distinctness. This muffled sound may be taken as the normal vocal resonance over a mass of normal lung tissue. This sound is not by any means the same all over the thorax. It varies much in intensity. In certain areas it becomes much more distinct, approaching in clearness and distinctness the tracheal sound. These areas are over the bronchi and at the inner part of

the supra-clavicular areas in front, and around the upper two or three dorsal and lower cervical vertebra.

The sound in these areas is so clear and distinct, when compared with that obtained over other areas of the lung, that the term bronchophony is applied to it. Gee defines bronchophony as "the clearest resonance ever heard over the healthy chest, and all degrees of clearness greater than this." Thus in a normal chest we can have muffled vocal resonance and bronchophony. In women and children and stout patients the vocal resonance is greatly diminished over the greater part of the chest.

Vocal resonance may vary in intensity and quality. (1) It is diminished, as a rule, in cases of pleural effusion, and this furnishes a most valuable sign. It is also diminished in cases of œdema of the lung; in obstruction of a large bronchus by an aneurism or tumour; occasionally also in pneumothorax and fibroid disease of the lung, and, very occasionally, in pneumonia. (2) The vocal resonance is increased in consolidation of the lung, as in pneumonia or in tuberculosis, in marked cases of emphysema in hæmorrhagic infarcts, in bronchiectasis, and in cavity formation.

In cases of pleural effusion a striking alteration of the vocal resonance is sometimes heard over the fluid in the neighbourhood of the angle of the scapula. The sound takes a nasal quality, and to this is applied the term **Ægophony**. The explanation of the phenomenon is obscure, though it is probably due to the interception, by the fluid, of part of the fundamental tones, while the over tones are little affected. Another variation in quality is sometimes heard when listening over a large cavity. A particularly hollow or echoing character is added to the sound. This is known as *Amphoric Resonance*.

**Adventitious Sounds.**—In addition to the breath sounds and the vocal resonance, there remain to be discussed what have been called adventitious sounds. These are sounds produced in the chest by disease. They are called *râles* by some, and by others crepitations. It will simplify matters if it is remembered that *râle* is another word for rattle, and crepitation is a synonym for crackle; so that when a listener states that he hears some *râles* or some crepitations,

he means that he hears some rattling or some crackling sounds in the chest. Some observers retain the word *râle* to describe intra-pulmonary sounds, as distinguished from extra-pulmonary sounds, such as these of pleural friction. Others again use the word *crepitations* so as to cover extra- and intra-pulmonary sounds. To make the nomenclature, which is somewhat confusing, more distinct, it may be well to put the terms in the following form.

*Râles or Crepitations.*

A. Moist . . . . .	{	Fine.
	{	Medium.
	{	Coarse or Bubbling.
B. Dry . . . . .	{	Sibilant rhonchi.
	{	Sonorous rhonchi.

It will help further if these are put side by side with Laennec's original classification :—

*Râles or Crepitations.*

1. Crepitant râles.	} Moist.	{ Fine crepitations or râles.
2. Subcrepitant.		{ Medium.
3. Mucous râles.		{ Coarse or bubbling crepitations or râles.
4. Sonorous and sibilant râles.	} Dry.	{ Rhonchi or sonorous rhonchi.
		{ Sibulations or sibilant rhonchi.

*Fine crepitation*, or, as it is sometimes called, fine hair crepitation, is a sound which is usually compared with that heard when a lock of hair is rubbed between the fingers close to the ear. It is heard in the first stage of pneumonia, cedema, or hypostatic congestion of the lung. It is heard during inspiration, usually toward the end. It is often extremely difficult to distinguish this from fine pleural friction. Fine crepitation is usually produced by the separation of the walls of the air vesicles, which are loosely adherent owing to the presence of some slight secretion, and when the air is drawn in during inspiration the sound is produced by the separation of these surfaces. *Medium crepitations* are caused by the air bubbling through fluid

secretion in the smaller bronchioles. They are found, as a rule, at the end of inspiration and at the beginning of expiration. *Coarse crepitations* are caused by the air bubbling through fluid secretion in the large bronchi. The mechanism of production in these two forms of crepitation is identical, the difference being in the place where the sound originates.

At times the crepitations are non-resonant or toneless, but at other times they have a definite resonance added, and are spoken of as resonant. This latter quality is usually found in the neighbourhood of a consolidated patch, or in the interior of a cavity. When a cavity is of some considerable size, with smooth lining walls, the crepitations in it or in its immediate proximity become high in pitch, and are spoken of as metallic or tinkling. This sound has been likened to drops of water falling into a hollow metallic vessel, which echoes with the sound of the falling drop. This tinkling crepitation is pathognomonic of a large cavity, or a pneumothorax, and may or may not be associated with the other signs of cavity, such as amphoric breathing. Even should all other signs be absent, well-marked tinkling crepitation is sufficient to justify a diagnosis of cavity. This is heard not infrequently in advanced phthisis.

*Dry Râles.*—These are often described as “snoring, cooing, or whistling” sounds. They are due to partial obstruction of the lumen of the air passages, usually by tough mucus. The pitch varies considerably. The high-pitched are termed sibilant rhonchi, and the low-pitched sonorous rhonchi, or more commonly simply sibilations and rhonchi. These sounds are usually found in bronchitis and catarrhal conditions of the lung. It must not, however, be assumed that their presence is confined to bronchitis, as many cases of early tuberculosis will show no other sign than a few persistent rhonchi or sibilations at the apex.

*Sounds simulating Râles or Crepitations.*—In certain patients the skin is harsh and dry, so that if there is the slightest movement of the stethoscope on the chest a sound is produced which simulates intra-pulmonary crepitations very closely. After applying a little vaseline to the skin the true nature of these sounds is detected, as they then dis-



appear. In a chest covered by hairs it is often impossible to decide the presence or absence of pulmonary râles, owing to the amount of crepitations caused by the stethoscope on the hair of the chest; but again a supply of vaseline applied to the skin of the part under auscultation corrects this. The swallowing of saliva produces a crepitation-like sound, which is heard over the apices during auscultation, but careful observation will, of course, detect the origin of these sounds.

At the apices certain indefinite creaking sounds are at times heard, which have been attributed to the presence of pleural adhesions or fibroid changes in the lungs. They are not often met with, and it seems quite reasonable to suppose that dense fibrous tissue should be capable of producing such creaking sounds. In the supra-spinous fossa there is very occasionally heard what has been called shoulder-joint friction. The sound is of a dry, friction-like character, and increases in intensity as the stethoscope approaches the shoulder joint. Such a sound is of very rare occurrence. Occasionally in muscular patients some sounds are heard that are muffled and rumbling in character. These appear to be due to muscular contractions in the chest muscles and need not be confused with intra-pulmonary sounds.

**Pleural Sounds.**—In health the pleura produces no sound, but in disease a certain characteristic sound is produced, namely friction. This is heard best in cases of pleurisy when fluid is absent, or not in sufficient quantity to separate the inflamed surfaces. As a rule it is of a very distinct, easily distinguishable rubbing character, but at times it is quite impossible to state whether the sound is friction or intra-pulmonary crepitation. Friction may be fine, medium, or coarse.

The distinguishing points about it are—(1) its rubbing character; (2) its rhythm. It may accompany both inspiration and expiration, and if so, this distinguishes it from the greater number of intra-pulmonary sounds, as the majority of those likely to be confused with friction are heard only during inspiration. At the same time it occasionally happens that the friction is only heard during part of the respiratory act, or only during a part of inspira-

tion, and this adds to the difficulty in recognising it. At times, when the inflamed pleura is close to the heart, the sound partakes of the "to-and-fro" character which is usually associated with pericardial friction. Coughing has little or no modifying power on friction, whereas it may have a very decided effect on crepitations and rhonchi. Another sound to be noted in connection with the pleura is that of Hippocratic succussion. This is pathognomonic of a large cavity in which there is both air and fluid (hydro-pneumothorax). The splashing is produced by shaking the patient suddenly, and is heard by applying the ear directly to the chest wall. When this sound is once heard it cannot again be mistaken.

## CHAPTER XIII

### SPECIAL TESTS

IN addition to the ordinary physical examination of the patient, certain other tests are available for assisting still further the diagnosis.

The value of these tests, and the dependence placed on them, are usually in inverse ratio to the experience of the examiner and his expertness in the ordinary physical examination of the chest.

**Sputum Albumen Reactions.**—It is an old observation that the sputum in many cases of tuberculosis contains albumen. This can be tested for by the ordinary nitric acid test or, better still, by the following method. Sputum which has been expelled from the lungs is taken, mixed with three or four times its bulk of normal saline solution, acidulated with a few drops of acetic acid, and shaken until it is homogeneous. It is then strained through filter paper, and the clear filtrate tested by boiling. When the lung tissue is damaged, albumen is almost always present. It is absent in bronchitis, so the test serves to distinguish bronchitis from tuberculosis. Unfortunately, however, as the test is positive in cases of bronchiectasis and abscess of lung, and is frequently negative in the very early stages of tuberculosis, its practical value is much impaired.

**Exercise or Auto-inoculation Test.**—In a healthy person exercise increases the temperature, but it speedily returns to the normal on resting a short time. In the tuberculous patient the temperature is more unstable, more readily deranged, and returns more slowly to the normal after exercise. In carrying out this test the temperature of the patient is taken carefully before he begins his exercise. He is then given a distance of from two to three miles to

walk and the temperature taken on his return. Such a walk may raise the temperature two or more degrees, but in the healthy individual this will fall again very shortly. In the tuberculous patient, however, the fever will last well over half an hour, and in some cases two hours, so that it is customary to take the temperature immediately on returning and half an hour afterwards. There is now much scepticism as to the worth of this test. Too many uncertain factors enter into it, and these make it of no value. There is the general condition of the patient; whether he is accustomed to walking or not; the rate at which he walks; the temperament of the individual; the place in which he rests on return, whether in a warm room or on a balcony, and the amount of clothes he wears when resting. Each of these factors has a considerable influence on the temperature, and in the writer's judgment the test is useless.

**Opsonic Index Test.**—This was introduced by Wright in order to estimate the amount of opsonins normally in the body or developed by the injection of tuberculin. It was further applied as a test of the amount of auto-inoculation produced by exercise or graduated labour. The technique was delicate and laborious, and the results arrived at with difficulty. The aim was to find out how many bacilli were ingested by the polymorphonuclear cells when these were incubated with an emulsion of the tubercle bacilli. It was a very pretty test, but after prolonged personal experience with it the author discarded it, as the chances of error were too numerous for the test to be of the slightest value. It is rarely done now.

**Complement Fixation Test.**—An enormous amount of work has been done in order to find in tuberculosis a reaction analogous to the Wassermann test in syphilis, but so far without any decided results. One great difficulty appears to be the procuring of a suitable antigen. Various antigens have been tried—alcoholic extracts of specially grown bacilli, mixtures of different alcoholic extracts, dead bacilli and living bacilli—but with unsatisfactory results.

It is quite probable that we may never get any more definite test than the cutaneous test to be discussed shortly, as in tuberculosis the question does not turn upon infection

or non-infection, but rather on (a) infection plus active disease, or (b) infection without active disease. We require a laboratory test which will enable us to distinguish between the individual who is merely infected and the individual who is suffering from active tuberculosis, and this we have not yet got.

**Tuberculin Tests.**—Tuberculin has been used in a variety of ways as a test for tuberculosis. It has been applied to the conjunctiva, to the skin, and it has been injected subcutaneously. Thus we have the ophthalmic test, the various cutaneous and the subcutaneous tests. The *Ophthalmic* test has been associated with the names of Calmette and Wolff Eisner. It is performed as follows: One drop of a one per cent. solution of old tuberculin is instilled into the conjunctival sac of one eye, the eyelid being held down for a short period in order to ensure that the tuberculin is not immediately pressed out. In a tuberculous subject, within six to ten hours the conjunctiva becomes injected, and there is lachrymation and some slight photophobia. The patient may complain of a gritty feeling in the eye affected. The redness of the eye reaches its maximum in about twenty-four hours and then slowly subsides. The test is a delicate one, and the slightest reaction can be readily detected by comparing the two eyes. It is, however, not without its dangers. The author has seen conjunctival ulcerations result from this test which required many weeks to heal—and in his judgment this test should not be employed at all. Some other observers, however, state it may safely be employed when there is no pre-existing eye disease.

*The Cutaneous Tests.*—(a) The Von Pirquet cutaneous test is performed as follows: The skin of the forearm is cleansed with ether, and when dry a drop of old tuberculin is placed on it. The skin is scarified through the drop. The scarification should just reach the dermis and should not draw blood. Von Pirquet used a special scarifier which had a chisel-shaped end about  $\frac{1}{8}$  inch in size and moderately sharp. This end was placed on the skin, and by a rotatory movement of the instrument the abrasion was made. By using a scarifier of a definite size the similarity in size of all the abrasions is assured. The



tuberculin is left in contact, for about five minutes, with the wound, and then the excess of fluid mopped off. A dressing is not necessary. Another abrasion is made at the same time on the forearm about 2 inches from the first one, but no tuberculin is applied, and care is taken that there is no chance of its being contaminated with the tuberculin from the test inoculation. This acts as a control.

In a positive result there is a definite induration of the skin at the seat of inoculation, and the formation of a slight papule. This is surrounded by a circular area of inflammatory redness, the extent of inflammation varying with the severity of the reaction. The arm should be inspected at the end of twenty-four hours, and the test inoculation compared with the control. The reaction is at its height, as a rule, at the end of twenty-four hours, and then gradually subsides.

Various types of reaction have been described. Thus there is the *premature* reaction, which comes on in from four to six hours, reaches its maximum in about ten, and then disappears rapidly. The *persisting* reaction, instead of passing off in two or three days, may last one or two weeks before it subsides. The *late* reaction does not appear until about forty-eight hours after the test was made. This reaction and the preceding one are stated to be more common in inactive lesions. The *cachectic* reaction is one in which a definite skin infiltration takes place, but is not accompanied by the red inflammatory area. This type is said to take place in late stages of the disease. The *scrofulous* reaction is seen occasionally in delicate children. Numerous small nodules appear around the primary papule, and these run a course similar to the original abrasion.

The pathology of the papule is interesting. It has the histological structure of a tubercle. There is an extensive round cell infiltration with epithelioid giant cells of the Langhans type.

(b) The intradermal test has been brought forward by Mantoux. It is simply a modification of the ordinary skin test. It is done by injecting, by means of a very fine needle, a small drop of tuberculin into the skin tissue. The needle is forced into the skin at an acute angle so that it traverses a considerable distance to get into the substance of the dermis.

The solution of tuberculin is then slowly pressed out so as to make a small white wheal on the skin. This is the sign that the tuberculin has been injected into the skin and not under it. A positive reaction under this test is similar to that of Von Pirquet's method.

An attempt has been made by this test to estimate the degree of hypersensitiveness to tuberculin which the patient possesses. Various dilutions of tuberculin are used, and  $\frac{1}{20}$  c.c. of each is injected separately into the skin, the weakest being used first.

The strength of the various solutions and the dosage are as follows:—

$\frac{1}{20}$  c.c. of 1 to 1,000,000 tuberculin dilution.

„ 1 „ 100,000 „ „

„ 1 „ 10,000 „ „

Each dose is injected into the skin of the forearm about 2 inches from the others, care being taken to have the syringe thoroughly cleansed after each injection. If these do not react, the other arm is used with the following stronger dilutions:—

$\frac{1}{20}$  c.c. of 1 to 1000 tuberculin dilution.

„ 1 „ 100 „ „

Romer is of opinion that the degree of reaction runs parallel with the severity of the injection, that is, the more active the disease, the weaker the solution required to cause a reaction. As a diagnostic test the intradermal has no value over the cutaneous one.

(c) Moro's test is also a modification of the cutaneous test. Moro's ointment, which is employed for the test, consists of equal parts of old tuberculin and lanolin. A small piece of the ointment about the size of a pea is used, and rubbed into an area of unbroken skin about 2 inches square on the surface of the abdomen. If the test is positive, in from twelve to forty-eight hours a crop of small pustules appears which persists for some days. They gradually subside in from five to ten days. This test is not so reliable as the others.

*Subcutaneous Test.*—This was the first tuberculin test we

had, and it was introduced by Koch himself. His original tuberculin is used, diluted with a normal saline solution, so that doses of 0.001 c.c. can be conveniently administered. The method of dilution is dealt with in the chapter on Treatment. Koch's own procedure was to give three separate doses as follows:—The first dose was 0.001 c.c. (1 mg.); two days later, if no reaction occurred, a second dose, 0.005 c.c. (5 mg.), was given, and if no reaction was manifest in about three days, a third maximum dose of 0.01 c.c. (10 mg.) was injected. If still no reaction was manifest, in five days the dose of 0.01 c.c. was repeated.

This dosage was employed for many years, but later observers modified it somewhat. The initial dose now used is often smaller than 0.001 c.c., and the maximum as stated by some should be 0.005 c.c. These doses, it should be observed, both minimum and maximum, are quite arbitrary. Löwenstein, for instance, states he gets good results by giving  $\frac{1}{2}$  mg. doses every second day for about a week. For ill-nourished, debilitated patients smaller doses should be employed, and 5 mg. should not be exceeded. For children, doses of from  $\frac{1}{10}$  mg. to a maximum of 1 mg. should be used. The injection should be given in the morning, as there is a better chance of noting any slight early rise of temperature. If given in the afternoon or evening, there is always the chance of a reaction occurring during sleep and thus being missed. The injection is usually made in the loose subcutaneous tissue between the shoulders or in the back of the upper arm. The former region is to be preferred, as, not infrequently, an œdematous swelling appears in the upper arm after a dose of tuberculin, whereas this never occurs in the interscapular area. As the test is not without some risk, it should not be employed in routine fashion. Some patients have been made much worse by this test, and their downward progress began from the date on which it was applied. It should therefore not be employed where the diagnosis can be established by other means, as, for example, by the presence of bacilli in the sputum. It should not be employed where the patient is in a febrile condition, as this will mask any slight reaction which may occur. Again, it should not be used in cases

of recent hæmoptysis, as the reaction may produce a recurrence of the hæmorrhage.

Before a diagnostic test is employed a careful four-hourly chart should be kept of the patient's temperature for a period of four days. This should preferably be a rectal temperature. The necessity for this preliminary observation is to ensure that there is no fever at the time the test is being applied. After it has been made, similar observations should be continued, preferably at two-hourly intervals, during the waking hours. By recording the temperature thus frequently, slight temporary rises in it are not overlooked.

When the test is positive the patient is said to have had a reaction or to have reacted to tuberculin. This reaction may be (1) general, (2) local, or (3) focal.

The **general reaction** appears within six to twelve hours, and as a rule is over in twenty-four to thirty-six hours unless it is particularly severe. Sometimes it begins as early as four hours after the injection, or it may be delayed for thirty-six hours. The onset may be sudden, with chill or rigor. Usually there is malaise, headache, and slight shivering. There may be pains in the back and limbs. These symptoms vary in severity. At times they are particularly severe, and may be accompanied by a marked increase of the cough and blood-stained sputum. The important point in the reaction is the fever. A rise of at least one degree Fahrenheit should be demanded if the general symptoms are slight. A rise to 100° F. would be reckoned a mild reaction. Up to 102° F. might be described as moderately severe, and above 102° F. as severe. The slight degrees of fever are a little disconcerting. When there is any doubt as to the cause, a second dose should be given. This usually settles the point, as the reaction will be quite definite then.

**Local Reaction.**—This consists of redness, swelling, and some degree of tenderness at the site of the injection. The swelling varies in size and may cover, in an extreme case, several square inches. The infiltration has some of the histological features of a tuberculous infiltration. The regional glands are usually slightly swollen and tender.

The injection of tuberculin in sufficient quantity produces an area of inflammation around each tuberculous nodule.

This is well seen in the case of lupus when, after injection, the healthy area of skin surrounding the lupus patch becomes red and congested. It is presumed that a similar change takes place in the lung tissue, and this is spoken of as the **focal reaction**. This change is recognised clinically in three ways: (*a*) by increased crepitations at the site of the lesion, (*b*) or by crepitations appearing at a spot in the lung where prior to the injection they were not audible, or (*c*) by an increase in the extent of the dull area. The focal reaction is often most disappointing and of little or no assistance in coming to a definite conclusion in any given case; frequently no crepitations are noted nor any extension of the area of dullness. Different observers have obtained widely different results in the observations of these focal reactions, the percentage of cases in which reaction was noted varying from 10 per cent. to 80 per cent.

If a patient has been tested previously by the ophthalmic or cutaneous methods, a second reaction may take place after the subcutaneous injection of tuberculin. These secondary reactions may occur as long as four or five months after the primary test. It is stated that they occur, at times, even when the first test appeared to be negative. Such reactions are of no clinical importance.

**Value of Tuberculin Tests.**—A reaction means an infection and nothing more. In the early days of these tests, before their limitations were fully appreciated, much needless worry was often caused by a diagnosis of tuberculosis being made, the sole basis for the diagnosis being a positive tuberculin test. Patients have been notified to the Public Health Authorities, and a request for institutional treatment made simply on this ground alone. Now we know better.

It has been asserted by some observers that the degree of sensitiveness—or as it is sometimes termed hypersensitiveness—exhibited by a patient corresponds to the degree of activity of the disease. The more active the lesion the smaller is the dose of tuberculin required to cause the reaction.

The degree of sensitiveness does not, however, vary uniformly with the extent or activity of the disease, nor



indeed, for that matter, even between active and inactive disease, so that there is no dividing line that will enable us to state, in any given case, whether a lesion in an apex is healed, quiescent, or active. A negative reaction is of much more value than a positive one. In the case of the cutaneous reaction the test should be performed at least twice before a negative result is accepted. If negative on two occasions this would wellnigh exclude tuberculosis. A most important exception to this is that no reaction may take place in far advanced cases of the disease. This is apparently due to the fact that the tissues are so exhausted or so soaked in tuberculin of the patient's own production that they fail to respond to the test dose introduced. Again, after measles, some cases of whooping-cough, and some of the other specific infectious diseases, the cutaneous reactions will be temporarily negative. This is supposed to be due to exhaustion of the tissue cells after these diseases, so that they are unable to create the antibodies necessary for the production of a reaction, or that the antibodies when created have been destroyed by the toxins of the previous illness.

No reaction takes place in the newly-born child. This fact in former times gave rise to curious speculation as to the cause. It was assumed to be due to the more active metabolism in the child as compared with a healthy adult, who although healthy reacted to the test. The real explanation, of course, is that the newly-born child does not react because it has not yet been infected with tuberculosis. The sum total of all tuberculin tests is, that with growing experience, one is less and less inclined to attach much importance to their diagnostic value.

## CHAPTER XIV

### X-RAY EXAMINATION

AN X-ray examination of the chest is a valuable addition to the routine procedure, but it cannot displace a careful physical examination. At times its help is invaluable: at other times it yields no information of importance. In the very early stages of the disease, for instance, it often proves to be of no help, yet an examination of the chest should not be regarded as complete until it has been radiographed. In order to appreciate the abnormal, it is essential that the student should be familiar with the normal appearance of the thorax under X-ray examination.

The chest may be examined by screening or by photograph. The latter can be studied leisurely and exhibits changes which are not perceptible on the screen. The photograph should be taken, if possible, instantaneously, as thereby the lack of definition caused by the movements of the heart are avoided. An examination by the screen has certain advantages over a plate. The movements of the diaphragm and ribs can be studied; the comparative illumination of the various parts of the lungs, and the changes brought about by respiratory movement, can be observed.

**Position of Patient.**—The patient can be most conveniently examined in the erect position, and then he can rapidly be screened in various positions, anterior, posterior, or oblique.

*Anterior Position.*—In the anterior or frontal position the patient stands with his face to the screen and his back exposed to the tube, and in this position a good view of the chest can be obtained. The first point which the observer notes is the central or median shadow, with the clear

pulmonary area on either side and bounded below by the diaphragm. The median shadow is made up of the sternum, heart, and great vessels, with the vertebral column posteriorly. The shape of the shadow is irregularly quadrilateral, or, at times, somewhat triangular, and varies according to the development and position of the heart.

The right border is made up of the superior vena cava and the right auricle. The left border is formed by the

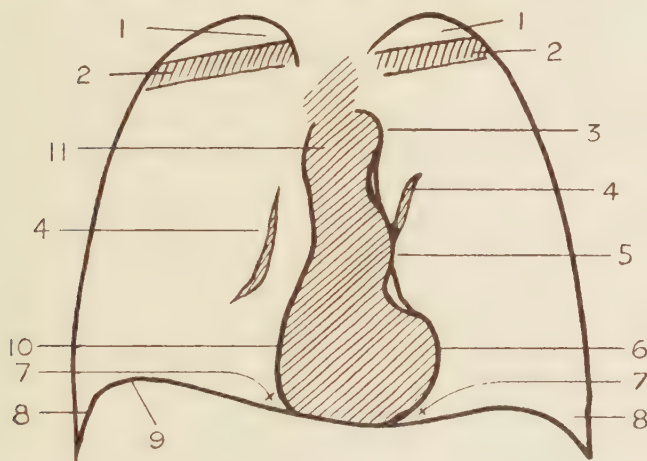


FIG. 32.—Diagrammatic representation of X-Ray picture. Anterior position.

- |                      |                                  |
|----------------------|----------------------------------|
| 1. Apices of lung.   | 7. Cardio-diaphragmatic sinuses. |
| 2. Clavicles.        | 8. Costo-diaphragmatic sinuses.  |
| 3. Arch of aorta.    | 9. Diaphragm.                    |
| 4. Hilus shadow      | 10. Right auricle.               |
| 5. Pulmonary artery. | 11. Superior vena cava.          |
| 6. Left ventricle.   |                                  |

aortic arch, the pulmonary artery, and the left ventricle. On either side of the median shadow can be seen the hilus shadow. On the left side this is much obscured by the heart, but it is well seen on the right side. The shadow is slightly crescentic in shape and projects downwards towards the diaphragm. The cause of the shadow has been much disputed, but it is now thought to be mainly due to the pulmonary arteries and veins. The bronchi in the normal condition do not contribute much to it. When the hilus shadow is abnormally dense other factors come

into play, as enlarged or inflamed glands, bronchial and peribronchial fibrosis. These conditions are not necessarily the result of tuberculosis alone. They may be due to repeated attacks of bronchitis, to dusty occupations, etc.

The position and movement of the diaphragm shadow will next attract attention. This runs transversely just below the heart, and is slightly higher on the right side than on the left. It should move equally on both sides, although in many patients without apparent pathological reason it moves slightly less on the right side. At the junction of the heart shadow and the diaphragm shadow there is formed what is called the cardio-diaphragmatic sinus. Where the diaphragm meets the ribs it dips down and forms an acute angle known as the costo-diaphragmatic sinus. During inspiration the sinus lights up well and becomes very distinct, but is lessened in extent and in translucency during expiration. This angle is of importance in the diagnosis of cases of pleurisy, as it early becomes obliterated if there is effusion. There is also obliteration of the cardio-diaphragmatic sinus in pleuritic or pericarditic changes.

The clear lung area is divided into the apex, and the body of the lung by the shadow of the clavicle. Running obliquely across the clear space of the bulk of the lung are the rib shadows. The apices should be studied carefully. They are not so well illuminated as the remainder of the lungs, nor do they light up so well on inspiration. The fact that many people in inspiring deeply raise the clavicles further contributes to the loss of illumination. When such loss is slight and equal on both sides, little attention should be paid to it. This is especially so where the patient is stout or has well-developed shoulder muscles. A unilateral loss of translucency is of pathological origin. In females the breasts cast a diffuse rounded shadow on the lower portion of the lung areas.

*Posterior Position.*—When the patient has been examined in the anterior position and the various points of importance noted, he is then turned right round with his back to the screen. In this position it will be seen that the outline of the median shadow is slightly different from that obtained



FIG. 33.—Normal Thorax. Single flash exposure,  $\frac{1}{100}$  second.





in the anterior position. This is due to the fact that the heart and vessels are now further from the screen and the shadow is much increased. The slope of the ribs can be better examined in this position, and any narrowing of the interspaces on one or other side can be easily observed. In this position, shadows due to lesions on the posterior aspect of the lung or pleura can be better seen than in the anterior position. The movement of the diaphragm can be again studied, as also the appearance of the costo-diaphragmatic sinuses.

*The Oblique Position.*—There are numerous oblique positions, but that most commonly used is what is known as the right oblique. The patient is slowly turned from the anterior position until the vertical nipple line on the right side is in contact with the screen. In this position the dense vertebral shadow is seen on the left, and in front of it the aortic shadow, with a clear space between the two. Running across the middle of this clear space is an indistinct hilus shadow. This position gives a good view of the arch of the aorta, and the clear space should always be examined carefully in cases of suspected tuberculosis of the root glands.

In early tuberculosis, as already stated, the aid received from the X-ray examination is often negligible. In these cases the older means of examination, viz., percussion and auscultation, give more certain information. Nor is this to be wondered at. The X-ray picture is, after all, only the picture of a shadow. If the newly-formed tuberculous tissue in the lung in the early stage of the disease is not dense enough to create a shadow, it will be missed altogether at the examination. But even in early stages of the disease, when the physical signs are doubtful, an auxiliary examination should always be made by X-rays.

The apices should be closely examined. Theoretically they should illuminate equally, hence any apparent difference in lighting up should be carefully scrutinised. This loss of illumination, if it occur on the side suspected on physical examination, may be taken as confirmatory evidence of the presence of disease. An examination of the patient should always be made so as to exclude the possibility of this

haziness being due to some such condition as supraclavicular glands, alterations of the shape of the clavicle or scapula, or to slight scoliosis. This latter condition causes a crowding together of the ribs on the concave side, and naturally there is less penetration by the X-rays with resulting loss of illumination on that side.

When the shadow is dense it may be assumed that the disease has been present for some considerable period, and that a great amount of fibrosis has taken place. Indeed, such an X-ray picture, in the absence of other signs and symptoms suggesting active tuberculosis, might be interpreted as an arrested or a healed lesion. When, in addition to apical obscurity, it is found that the upper ribs are limited in movement and crowded together, it may safely be assumed that there is an old-standing lesion. It should be remembered that it is quite possible for the X-ray examination to reveal an old-arrested lesion in one apex and miss a recent active one in the other apex, and thus be quite at variance with other clinical findings. When both apices are equally obscure a difficult situation arises, and it is often impossible to say by X-ray examination whether the condition is pathological or not. In healthy individuals the apical illumination varies within wide limits. In thin patients the apices show up clear and distinct, whereas they may be quite indistinct and grey in stout people or those with well-developed shoulder muscles.

**Examination of Hilus.** — After the apex has been examined attention will be directed to the hilus shadow. This shadow is subject to wide variations, even in people clinically healthy, and those variations have given rise to many erroneous opinions. Many a diagnosis of tuberculosis has been made because of an increase in the hilus shadow, due to bronchial and peribronchial thickening of a non-tuberculous character. It should be clearly understood that any inflammatory process will increase the shadow in extent and density. It is increased, for instance, in simple or chronic bronchitic conditions, after measles or whooping-cough, and is invariably and permanently increased and intensified in those who work at a dusty occupation.

In early disease there may be some increase of the

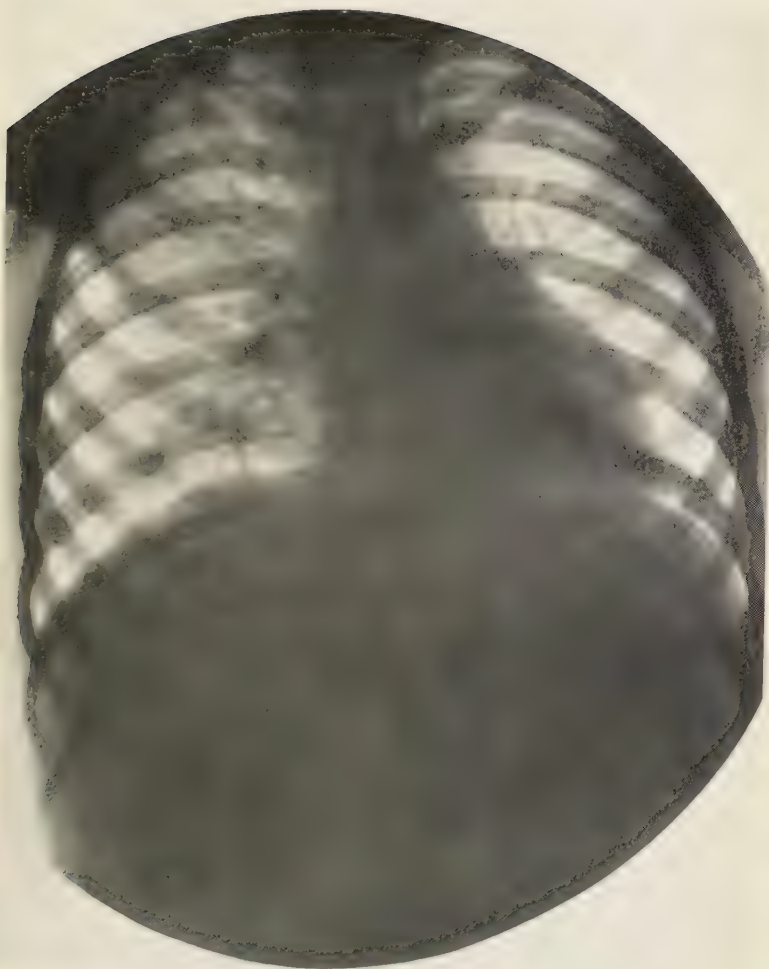


FIG. 34.—Tuberculosis of the Right Lung. Left also involved.





shadow on the affected side, and delicate strands may be noted running from the hilus up to the apex. These strands may be either fibrous tissue or thickened and congested bronchi. The presence of calcareous glands which show up as dense spots in the hilus shadow merely indicate old disease. They are no proof that the patient is, at the time of examination, suffering from tuberculosis, neither are they proof that he has recovered and is not now so suffering. In short, the detection of calcareous glands does not help us much, if at all, in the diagnosis.

**Movements of Diaphragm.**—After the apex and hilus shadows have been inspected the movements of the diaphragm should be studied. In health both domes, as a rule, move equally, but in diseased conditions movement on one or both sides may be more or less impaired.

In early tuberculosis, as was pointed out first by Williams, there is at times some interference with the mobility of the diaphragm on the affected side. The excursion may not be so great, and the movement may be more irregular or jerky. Different explanations have been put forward for this sign. It has been variously attributed to reflex action or diminished air entry, or to fibrous development in the lung. The author has not been impressed by the value of this sign, as in a majority of early cases it is absent.

It will thus be seen that X-ray examination can no more settle off-hand for us the question of early tuberculosis than can the older methods. The diagnosis of early cases of the disease remains as before a difficult matter, and one into which the element of the uncertain enters very largely. When the disease passes into the more advanced stages, an X-ray examination furnishes us with most valuable information. Although the disease is well marked and physical examination furnishes abundant evidences of its presence, yet the X-rays often reveal a much wider distribution of the disease processes than is suspected from percussion and auscultation. It is here that the worker who combines the physical and X-ray examinations has the advantage. He gets into the habit of reading into the physical signs meanings which are not apparent to one unaccustomed to X-ray work—just as the worker who

combines clinical and pathological studies has an advantage. The X-ray picture in marked disease varies very considerably. It may reveal a dense shadow in an apex or widely scattered foci throughout both lungs. It may show lessened movement or immobility on one side, with ribs crowded together, and with massive dense consolidation at the base. In short, the picture varies in every case according to the position, distribution, and severity of the disease.

Cavities may often be detected with ease, but frequently a cavity which gives the classical signs on examination by stethoscope is missed altogether on X-ray examination. Again, at other times radiography will show what appear to be typical cavities, and yet these are not present on post-mortem examination. X-ray diagnosis of cavities is therefore not always reliable. When visible, cavities appear as clear spaces with dark borders. They are seen more readily when large and superficial, and those near the front of the lung are best seen when examined in the anterior position. Those situated near the posterior surface are best seen when looked at from behind.

An X-ray examination helps to confirm the physical findings, but it gives no idea as to the activity of the lesion, although radiologists would have us believe otherwise. A reliable finding as to activity can only be arrived at by a wide survey of the general condition of the patient and of the signs and symptoms which he presents.



FIG. 35.—Advanced Tuberculosis.



## CHAPTER XV

### DIAGNOSIS

A CORRECT diagnosis in pulmonary tuberculosis is all-important. It may make all the difference between life and death; between a good recovery and protracted invalidism. In well-marked cases of the disease, the diagnosis is usually easily made, but even in apparently well-defined cases of disease, mistakes happen. Lung tumours, aneurisms, bronchiectatic cavities, etc., have frequently been diagnosed as tuberculosis, as the physical signs, on a superficial investigation of the case, all appeared to warrant such a conclusion. If mistakes can occur in what appear to be well-marked cases of pulmonary tuberculosis, it is apparent that the difficulties in forming a correct diagnosis must be infinitely greater in the earlier stages of the disease, and especially where physical signs are few or altogether absent. Early cases of pulmonary tuberculosis are, speaking generally, comparatively easily cured. This is shown by the post-mortem findings of several workers in cases of deaths from various causes, where healed tuberculous lesions were discovered in the lungs. Such cases had recovered spontaneously. Many ill-defined cases of illness are really slight attacks of pulmonary tuberculosis. Unfortunately, they are diagnosed sometimes as cases of influenza, while at other times the patient is said to be suffering from overwork, or is "run down," or "below par." Recovery from these slight febrile attacks is the rule, convalescence being hastened by means of a holiday, change of air, etc. It is not improbable that were such cases carefully examined, the true nature of the disease would be recognised. Practitioners should be on their guard when dealing with all ill-defined illnesses, so as to avoid, if possible, overlooking early tuberculosis. If the



case is not clear, it would be infinitely better that the patient should be told that he is suspected of having tuberculosis, and that he should take the necessary steps to combat this disease, rather than he should be put off with an indefinite diagnosis such as "below par," etc. In such an event, the patient may return at a later date with well-marked physical signs of the disease.

The earlier the disease is detected, the greater is the chance of complete recovery. Unfortunately, however, the earlier the stage the greater is the difficulty in diagnosis. In many cases, nothing will tax the knowledge and experience of the physician more than the diagnosing of early tuberculosis.

The detection of tubercle bacilli in the sputum is the only certain proof that a patient has pulmonary tuberculosis. Other signs and symptoms may, by their collective presence, make a strong chain of evidence in favour of the presence of tuberculosis, but the completion of such evidence is the finding of the bacilli. Their presence, however, means that destructive processes have begun in the lung; that the lung tissue is disintegrating, and that there is constant danger of new areas of lung becoming affected. A patient with tubercle bacilli in the sputum has already passed the incipient stage of the disease, and the lung is breaking down, although clinically he may still be classified as Stage I. A diagnosis of pulmonary tuberculosis should always be made, if possible, before the lung tissue begins to break down, and it is extremely bad practice to wait until the tubercle bacilli are discovered before taking action. The difficulties of diagnosis in the early and more curable stage are great, as already indicated, and the practitioner must be prepared to spend time, and to exercise thought, in the process of forming a judgment.

There is no easy road to a correct diagnosis. The various tuberculin reactions, speaking in general terms, only prove a tuberculous infection, which is a totally different matter from clinical tuberculosis. The practitioner is not specially interested in normal individuals who have been, at some period of life, infected by the tubercle bacillus; his special interest lies in those who are actually suffering from the

effects of such infection. It is extremely difficult to draw a well-defined line of demarcation showing when infection has passed on to actual disease. For instance, a patient presents himself for advice and states that he is more easily tired than usual, that he suspects he has lost a little weight but is not very sure on this point. There is no cough, as far as he has noted, but he just feels run down. He explains that he thinks that his condition is due to overwork and lack of holiday. Physical examination of the lungs reveals nothing very definite—no crepitations, no consolidation—but the patient gives a positive reaction to the cutaneous tuberculin test. Is this patient suffering from overwork in, possibly, an unhealthy environment, or is he suffering from tuberculosis in an early stage? It is often very difficult to answer this question, and yet such a case is but typical of many. These patients present problems which even the most experienced physicians may fail in finding the correct solution. It is, of course, an easy matter to give an emphatic diagnosis of early pulmonary tuberculosis in all such cases, but to do this is open to grave objection. If the case were one of a suspected fractured rib, little or no harm would be done if a definite diagnosis of fracture were made and appropriate treatment applied, but a fractured rib is not tuberculosis. If a diagnosis of tuberculosis is wrongly made in any suspected case, it means much unnecessary mental anxiety to the patient and his friends, much unnecessary financial and business worry, and often some ostracism when work is resumed. Once a patient has been labelled by his associates "consumptive," a stigma is oft-times attached to him which is not likely to be shortly removed.

It is to be feared that there is a temptation on the part of tuberculosis experts to make an absolute diagnosis of tuberculosis in all these difficult cases. To yield to this temptation is as easy as it is dishonest. Such a diagnosis is no doubt the more readily made because it is very difficult for another observer to prove it to be wrong. Should the patient develop tuberculosis at a later date, great credit can be gained for having made a diagnosis at such an early stage. If the patient makes a good recovery, more credit can again be gained by announcing that he was just "got in time," and

his case swells the number of cures which are claimed on behalf of any special line of treatment which was adopted.

No cases of tuberculosis are so easily or permanently cured as those in which it never existed!

The correct method of dealing with these borderland cases is to state quite frankly that suspicions are entertained of tuberculosis, but that no definite diseased area can be detected. The patient ought to be kept under strict medical supervision, and should carry out, thoroughly, all reasonable hygienic methods of living. He need not be sent to a sanatorium, but the sanatorium should be brought to the patient, *i.e.*, his home should be his sanatorium. Later on, a short term of residence in the country or at the seaside usually completes and consolidates recovery. These suspicious cases should not, of course, be notified to the Public Health Authority.

It should be carefully noted that the above line of treatment is recommended in the case of patients without definite physical signs. Where a patient has some well-recognised tuberculous lesion in the lung, it is wrong to speak to him, or his friends, of a "weak spot in the chest," or a "delicate lung," or to use any other such meaningless term. A definite diagnosis of tuberculosis or consumption should be given even at the risk of causing some alarm. No attempt should be made to cover up the seriousness of the position, as at least 50 per cent. of these well-marked cases are dead within five years. Appropriate treatment should be begun at once and no procrastination allowed. It is to be feared that in the past too little attention has been paid in the medical training schools to the diagnosis of early tuberculosis. This has been due to two things: first, to a probable want of appreciation on the teacher's part of the early manifestations of the disease, and second, to the lack of clinical material on which to demonstrate to the students. Now, because of an exaggerated fear of infection, few Infirmary managers care to admit cases of tuberculosis to their wards, and those which are admitted are usually in an advanced stage of the disease. These, on being shown to the students as cases of tuberculosis, give a totally wrong impression of the disease. In order to see tuberculosis in its

early stage, students must be sent for a course of training to a tuberculosis dispensary or to a sanatorium. It would be well for the future race of practitioners if this were made a compulsory part of their training.

Seeing, then, that the difficulties of diagnosis in an early stage are great, and that it is all-important to make a correct diagnosis in spite of these difficulties, how can this be accomplished with reasonable certainty and with the minimum amount of error? It has to be stated that the only method is by painstaking inquiry into the patient's history, by careful physical examination, including X-ray examination, and supplementing these by other special tests. When this has been accomplished the judgment which is formed will depend greatly on the experience, clinical aptness, and power of interpretation and deduction which the physician possesses.

The discovery of a lesion in the lung does not necessarily prove the patient to be suffering from active tuberculosis. The problem has still to be settled as to whether this lesion is in an active or a quiescent condition. Is it a healed lesion or a commencing one? In an attempt to help in the solution of these problems a critical examination will now be made of all points relating thereto.

In cases where there is no sputum, or where, if sputum is present, an examination has shown it to be free from tubercle bacilli, the diagnosis has to be made on the evidence of constitutional or systemic disturbance, combined with the result of the physical examination of the lung. Attempts have been made to evaluate the relative importance of the constitutional symptoms as against the results of physical examination. Such attempts are quite useless, as one is complementary to the other. No matter how well marked constitutional signs are, they must needs be confirmed by physical examination.

The classical symptoms are cough and spit, loss of weight, loss of strength, night sweats, fever and hæmoptysis, hoarseness of voice, anæmia, and in an advanced case of the disease all of these may be present, and present in a marked degree. In the early stages, however, many of them are absent and others only just discernible, and this makes the

picture incomplete and renders the diagnosis difficult. In any one case there may be present only very slight cough and no sputum, some loss of flesh, but nothing more. In another there may be some slight loss of strength, diminution of weight, but no noticeable cough. In still another the patient may have been feeling perfectly well, but suddenly has had a slight hæmoptysis, and shows no other evidence of disease. Any combination of these symptoms or any single one of them may bring the patient for examination, and a considerable amount of time has to be spent in determining the physical signs in the chest.

Careful inspection should be made for any retraction or flattening above or below the clavicle. If found, and if present in a marked degree, it means that the lesion is not a recent one; but if constitutional signs are present, one is tempted to surmise that there has been a recrudescence of activity in the old lesion, though it may be that these symptoms are due to an entirely new focus of disease. A slight hollowing is more compatible with early disease, although such hollowing means that the disease has probably been present from at least eight to twelve months. In many early cases of the disease it should be clearly noted that there may be no alteration in the shape of the chest. As previously mentioned, the flattening may be due to scoliosis, and, before proceeding further with the examination, the spinal column should be carefully inspected. It may also be due to atrophy of muscle, which will be dealt with under Palpation.

Drooping of the shoulder is not found in early cases. This, if due to lung disease, is a late manifestation. If the hollowing is bilateral, this may mean an old fibrotic condition in both apices, and is not due to early tuberculosis. A general flattening of the upper part of the chest from the clavicles to the third or fourth rib is often met with, but this is not a sign of early tuberculosis. It is due rather to deficient development or to old fibrotic changes in the lung. The hilus dimple, a term which has been applied to a slight, circular depression about the inner part of the second interspace, is a sign of old-standing disease which has undergone cicatrization.

In early disease there may be on inspiration on the





FIG. 36.—Chest showing the flattening of Advanced Tuberculosis.



affected side either (*a*) a simple lagging behind the other side of the chest, while the ultimate excursion of both sides may be equal, or (*b*) a definite limitation in the extent of movement. This lagging or limitation of movement is due to deficient expansion of the apex, and this, in turn, may be due to (1) slight consolidation of the lung itself; (2) actual cicatrization of the lung; (3) tonic spasm of the muscles overlying the area.

Pronounced deficiency in movement or complete immobility are not signs of early tuberculosis. They are due to extensive fibrotic changes in the lung. Such cases, however, are often discovered on the first examination of the patient, who may be unaware of any previous illness. This, however, will not deceive the examiner, and a diagnosis of early tuberculosis should not be made. Limitation of movement at the lower costal margins of a lung should not be looked on as evidence of early tuberculosis.

Some patients are met with who show a considerable degree of deficiency of movement in both apices. This may be a sign of early tubercle in both apices, or of old fibrotic conditions. It may also be due to the fact that the patient is a poor, debilitated individual who never has thoroughly inflated his lungs, and has neglected physical exercise throughout life. The fact of the bilateral deficiency should be noted, and its value appraised at the end of the examination. Visible pulsation associated with retraction or diminution of movement in the second or third left interspace is a sign that the disease has passed the early stage, and that there is much cicatricial contraction.

A considerable amount of help is often gained by palpation. A patient may make no complaint of loss of weight, but the experienced clinician can often detect this at once by palpation. Marked emaciation even when the associated signs in the chest are trivial is a sign of advanced rather than early disease. Muscle spasm, to which attention has already been drawn, is a sign of early active disease, and should be detected, if present, at this stage of the examination. In some cases it is well marked, but care should always be taken to avoid confusing the apparent increased thickness of the muscle

with the hypertrophy due to occupation. When muscle degeneration and atrophy have supervened, the case is no longer in an early stage.

Vocal fremitus is not a point of much importance in early right apical disease, but is frequently of great value when the left apex alone is involved. The normal increase of fremitus in the right side varies within such wide limits that even an experienced observer may have great difficulty in stating when the increase has attained such a disproportion as to constitute a sign of disease. In the left apex, however, it is a different matter. When the fremitus is greater in intensity than that on the right, or even when equal, it may be looked on as confirming, in decided fashion, other signs of underlying early disease. Cases are met with occasionally where the increased fremitus in the left side is the most prominent physical sign which can be detected, and this forms a decisive factor in the diagnosis.

On pressing the chest wall, one occasionally gets a feeling of increased resistance on the one side as compared with the other, but this is not a sign of early tuberculosis, as, in order to get this resistance, there must be extensive consolidation of the lung. The significance and importance of the supra-clavicular glandular enlargement has already been dealt with.

Only light percussion should be employed in any case of suspected early tuberculosis. Comparative percussion, *i.e.*, comparing the note obtained over one apex with that of the other, is of most value. The relative sizes of Krönig's areas and the amount of tidal expansion are of importance; but these take only a second place, and comparative percussion comes first.

In early tuberculosis only slight impairment of the percussion note or slight alteration in pitch should be expected. Marked dullness extending to, say, the third rib does not mean early disease in any sense of the term—it has now become advanced. Basal dullness does not indicate early tuberculosis; if due to tubercle, it signifies far advanced disease. The author has never been able to demonstrate the existence of the "reflex bands" of dullness, which Rivièrè finds running across the supra-scapular area in cases of early tuberculosis.

He has searched repeatedly for them, but has never satisfied himself that they were present.

There are times when the percussion note appears to be unsatisfactory in both apices, and then it is difficult on percussion alone to decide as to whether the impairment is due to bilateral disease, to deficient expansion in both apices, or to old, slight cicatricial conditions. The fact should be observed that the apical note is unsatisfactory, and the value assessed later, in conjunction with other evidence. Occasionally the note is so slightly altered that the examiner cannot thoroughly convince himself as to any actual definite change, and in these cases the other percussion tests referred to in the following paragraphs should be employed.

When the difference in the comparative note is definite and decided, there is no need to proceed further with percussion, as the presence of disease is beyond question; but when a doubt exists as to the equality of the notes, or where there is only a very slight alteration of the pitch, then a careful delineation of Krönig's resonant areas becomes necessary. It is not often that one finds a very decided alteration in the extent of this area when there is no definite difference in the comparative percussion notes of the apices, but at times a distinct diminution is detected. Sometimes it happens that the upper border is not quite so clearly defined on the affected side as on the other. An alteration in the note begins to be detected before the limit of the resonant area is reached: the note becomes slightly dull. When Krönig's area has been outlined it is well, also, to mark out the amount of tidal expansion on each side, as some confirmatory evidence may again be obtained indicative of a lesion in one apex or the other. A considerable amount of practice in percussion is necessary before the results of mapping out Krönig's area or the respiratory excursion can be relied on.

Another method by which we may get confirmatory evidence of an apical lesion is by percussing out the actual apices accurately by Goldscheider's method. This, in skilled hands, can be done fairly accurately, although the inner edge is difficult to define owing to the proximity of the trachea. Goldscheider percusses the apex with the finger bent at

right angle, as shown in Fig. 37; in this way he can get, he thinks, more easily between the divisions of the sternomastoid. The author finds that he obtains more accurate results by placing only the tip of the finger over the apex and percussing with extreme gentleness over the finger-nail. The delineation of the actual apex is not of much practical value unless by a very accomplished practitioner, as the chance of error is too great.

There is some divergence of opinion as to what is the earliest change in the breath sounds in incipient tuberculosis. Some observers have declared that the first respiratory change is that the murmur is weakened. Others, and with these I am in agreement, state that the inspiration becomes harsher, raised in pitch, and with expiration slightly prolonged.

The term "granular respiration" has been introduced by Grancher to describe a breath sound indicative of early disease, but this seems to introduce a needless addition to our vocabulary. If harsh inspiration and slightly prolonged expiration are found in the left apex, it is of much more significance than when found in the right, where this type of breathing may be found normally. In the right apex it may be broncho-vesicular, or even bronchial, and yet not due to disease. It is extremely difficult to appraise, correctly, these changes in the right apex, so that unless accompanied by other signs, especially some impairment of the percussion note, it is better not to attach too much importance to them. When harsh or broncho-vesicular breathing is found in both apices equally, and no diminution of the percussion note is present, it is more likely that it is due to emphysematous changes rather than to tuberculosis. Diminished respiratory murmur at one apex is more frequently due to a cicatricial condition than to a commencing lesion.

The presence of persistent crepitation in one or other apex indicates active tuberculosis, and is a most valuable sign. Persistent apical rhonchi have the same significance as crepitations. It is true that, at times, moist sounds may be detected in the apex in various cardiac conditions, and in emphysema, but further examination will reveal that the





FIG. 37.—Goldscheider's method of percussing the actual apex.



FIG. 38.—Position of the lung apex above the clavicle.



sounds are, probably, due to these other diseases. Occasionally in early tuberculosis there may be only one single crepitation heard: the clicking rôle of the older writers. At other times no crepitations can be detected until after the patient has been made to cough at the end of expiration, and then there is heard a perfect shower of fine crackles. This occurs not infrequently, so that no chest examination is complete without listening for post-tussic crepitations. It sometimes happens that the only sign, which can be detected, indicating active apical tuberculosis is pleural friction, so the significance of this will not be lost on the examiner.

Some observers have declared that percussion reveals the changes in the lung before auscultation does, but with this the author does not agree. He believes that auscultation enables the clinician to observe these changes first. Fortunately, however, it is not a question of percussion versus auscultation, but it is the combined result of inspection, palpation, percussion, and auscultation which enables us to settle the presence or absence of local lung changes.

**Activity of Lesion.**—Having determined the presence of a lesion in the apex, the next step is to decide as to whether it is progressive or non-progressive, active or inactive. With the minute care and attention which are now paid to pulmonary conditions, many alterations in the apices are discovered which formerly, in less exact days, would have been passed over, and there is now the danger of assuming that wherever a lesion is detected it must necessarily be active. It cannot be too clearly understood that no alterations in the breath sounds are indicative of an active lesion. There may be broncho-vesicular breathing; there may be pure bronchial breathing; there may even be cavernous breathing, and yet these afford no evidence of activity. In my own practice I have known a patient exhibit typical cavernous breathing, with all the classical signs of cavity formation, for wellnigh a score of years, and still, to all intents and purposes, he was quite well. The lesion was apparently quite healed.

Crepitations are the only signs of activity which the stethoscope reveals. When they are heard persistently at

the apex they indicate active tuberculosis, but if heard at the base of the lung they are more likely to be due to congestion or other non-tuberculous conditions. At times crepitations may be heard in the apex and yet no great constitutional disturbance be apparent. This does not mean that moisture in the lung is the result of a non-tuberculous process. It may rather be that the extent of the lesion and the degree of activity of the tuberculosis is not at present sufficient to produce much general systemic disturbance.

Absence of crepitations does not mean absence of activity in the lung lesion, and this fact must never be forgotten. It may simply mean that the lesion is too deep for the sounds to be detected, or that the movement of air in the diseased lung is too slight to cause crepitations. In some patients who are obviously dying, it is only on rare occasions that crepitations can be heard in the chest.

In the absence of crepitations, how then can the degree of activity in the lung be estimated? We have to rely solely on the amount of constitutional disturbance, and especially on fever, pulse rate, and the extent and rapidity of the loss of flesh. A lesion producing no constitutional disturbance may be assumed to be arrested provided no crepitations are detected at the site of the disease. So, when the general symptoms are indefinite, and there is no apparent constitutional disturbance, it is necessary to be satisfied as to the presence of crepitations before assuming that any slight local changes in physical signs indicate active tuberculosis. When constitutional symptoms, fever, loss of weight, cough, or night sweats are marked, then the presence of crepitations in the lung need not be insisted on, but minor alterations in the physical signs, such as slight retraction or harsh inspiratory murmur, apical cogwheel breathing, etc., would be accepted as evidence of the presence of active disease.

In any suspected case of early tuberculosis, then, the diagnosis is the combined result of a careful survey of the history of the patient, the constitutional symptoms and disturbance, together with the findings of the physical examination. In order to assist the practitioner further at this difficult point, and to show the result of the deliberations

of other workers in the field, there is appended the definitions and standards for diagnosis adopted by the American National Association for the study and prevention of tuberculosis :—

## DEFINITIONS.

1. *Loss of Weight*.—By “loss of weight” should be understood an unexplained loss of at least 5 per cent. below normal limits, for that particular individual, within four months’ time.

2. *Loss of Strength*.—By “loss of strength,” in its pathological sense, is meant undue fatigue and a lack of staying power which are unusual for the individual patient, and which cannot be satisfactorily explained.

3. *Fever*.—An occasional temperature of 99° F. should not be considered “fever.” A temperature which persistently runs over 99.4° F., when taken at least four times a day over a period of one week (by mouth five minutes), should be considered of significance and to constitute “fever.”

4. *Elevation of Pulse*.—Where the average normal pulse of the patient is already known, an elevation of 15 beats per minute, when the pulse is taken quietly at home during various periods of the day, should be considered abnormal. In cases where the average pulse is not known, and of course this constitutes the majority of cases, one should consider an average pulse of 85 or over in men, and 90 or over in women, to be abnormal. The combination of a subnormal temperature and an elevated pulse as defined here should be considered of great importance.

5. *Hæmorrhage*.—Any amount of expectorated blood, with or without sputum, may mean that tuberculosis is present and requires careful and thorough medical investigation as to its source. Blood streaks, blood spots, etc., may or may not mean tuberculosis. On the other hand, a hæmorrhage of one or two teaspoonsful is presumptive evidence of the disease.

6. *Family History*.—An occasional case of tuberculosis in the patient’s uncles, aunts, cousins, etc., should not be considered of importance, unless there has been intimate exposure and personal contact with such a case. It is an important fact when the patient’s immediate relatives, such as brothers, sisters, father, mother, or grandparents, have been tuberculous, and especially so when there has been prolonged and intimate contact.

7. *Exposure*.—Childhood exposure is of the greatest importance. Moderate exposure among normal healthy adults of cleanly habits is

of less importance. Of course, prolonged contact, with unhygienic habits or surroundings, may be a dangerous factor at any age.

8. *Cough*.—There is no cough characteristic of tuberculosis. Every cough that persists for six weeks or over requires investigation. Tuberculosis may exist without any cough whatsoever.

9. *Sputum*.—The presence of sputum is not necessary for a positive diagnosis. The constant raising of sputum, with or without cough, requires investigation. Absence of bacilli in the sputum after one or several laboratory examinations is not necessarily proof against the presence of active tuberculosis.

10. *Hoarseness*.—Any hoarseness or a persistent “huskiness” requires investigation.

#### MINIMUM STANDARDS.

On the basis of these definitions the following minimum standards in the diagnosis of pulmonary tuberculosis have been formulated.

1. When constitutional signs and symptoms and definite past history are absent, or nearly so, there should be demanded signs in the lungs, including persistent râles at one or both apices. By “persistent” it is meant that the râles must be present, after cough, at two or more examinations, the patient having been under observation at least one month.

2. In the presence of constitutional signs and symptoms, such as loss of weight and strength, etc., as defined above, there should be demanded some abnormality in the lungs, but not necessarily râles.

3. Usually a process at the apices should be considered tuberculous, and a process at the base to be non-tuberculous, until the contrary is proved, excepting when a clear history of pleurisy is present.

4. A hæmorrhage as defined above is evidence of active pulmonary tuberculosis until the contrary is proved.

5. One should consider a typical pleurisy with effusion as presumptive evidence of tuberculosis. One should also consider a dry pleurisy evidence of slight tuberculosis.

6. Pain in chest and shoulders, night sweats, digestive disorders, etc., may be present and should be investigated. Fistula in ano should be considered as a tuberculosis manifestation, requiring careful examination of the lungs for traces of the disease.

7. In every doubtful case one should demand that the patient be kept under observation for at least one month, with repeated sputum examinations, before a definite diagnosis is made.



## CHAPTER XVI

### TUBERCULOSIS IN CHILDREN

TUBERCULOSIS is not an infrequent cause of death in children, but it is the non-pulmonary forms which contribute chiefly to the death-rate. This is shown by the following table, which gives the Scottish death-rate at different ages from the various forms of the disease :—

SCOTLAND 1920.				
	Pulmonary Tuberculosis.	Tuberculous Meningitis.	Abdominal Tuberculosis.	Other Tuber- culous Diseases.
Under 1 year .	18	146	68	51
1- 5 years .	78	218	149	93
5-10 " .	72	102	72	63
10-15 " .	141	60	50	61
15-25 " .	1016	41	76	146
25-35 " .	976	13	38	94
35-45 " .	813	6	25	76
45-55 " .	631	4	22	61
55-65 " .	308	...	18	55
65-75 " .	117	...	6	21
75 and upwards	24	...	1	12
	4194	590	525	733

It will be seen that up to the age of fifteen pulmonary tuberculosis plays only a small part in the death-rate.

In young children the non-pulmonary forms of the disease predominate. In children under one year meningitis is the most common fatal form. The type of pulmonary tuberculosis with which we are so familiar in adults is not often seen in young children, although occasionally chronic

tuberculosis, of the adult type, with cavity formation, is met with. When tuberculosis attacks the lungs of infants and young children, if the dose is massive enough, the disease assumes the acute broncho-pneumonic or miliary types, and death is usually the result.

If the dose is insufficient to produce an acute general form of the disease, small local lesions are caused and these are not confined to the apex. These primary lesions, occurring as they do in any part of the lung, have often few or no localising signs. They may apparently heal, but the bacilli pass along the lymph channels and infect the glands which drain the affected area. Ghon maintains that the infection of the root glands is always secondary to a definite lesion of the lung. Others insist that in many cases the bacilli pass through the lung epithelium, as they do through that of the intestine, without causing any lesion, and that the primary focus is in a root gland. This is one way in which tuberculosis of the glands at the root of the lung is produced, but in other cases the bacilli are brought to the lung by the blood-stream, caught in the pulmonary capillary network, escape into the lymphatics, and are then conveyed by the lymph-stream to the root glands. Thus it is we have to study tuberculosis in children more frequently as an affection of the root glands rather than of the lung itself. The term *hilus phthisis* has been applied to this form of tuberculosis.

In order to grasp intelligently some of the signs and symptoms, the following brief summary is given of the arrangement and distribution of the root and tracheo-bronchial glands, for which we are indebted to Dickey's admirable thesis on *Applied Anatomy of Lungs and Pleural Membranes*.

**"Anterior Deep Cervical Glands.**—Bartels describes under this name two important groups of glands situated along the front and sides of the larynx and trachea.

"1. *Median Group*, situated in the middle line of the neck and divisible into two sets:—

"(a) Pre-laryngeal (1 or 2 in number), lying on the crico-thyroid membrane, or cricoid cartilage, and draining the larynx and thyroid body.

“(b) Pre-tracheal, accompanying the inferior thyroid veins and deriving lymph from the thyroid body. They are connected with the lateral tracheal and the superior mediastinal glands, and may be practically continuous with these two groups.

“2. *Lateral Group*.—These are situated along the course of the recurrent laryngeal nerve, in the groove between the trachea and œsophagus. They are continuous below with the tracheo-bronchial glands of the thoracic cavity. These glands when enlarged may lead to compression of the recurrent laryngeal nerve, and in this way laryngeal paralysis may be set up.

“The anterior deep cervical glands receive lymph from the larynx, trachea, pharynx, and thyroid body. They open into the jugular trunk, directly, or by way of the lower deep cervical glands.

“Their communications with the superior mediastinal glands (Bartels) and with the tracheo-bronchial glands (Beitzke and Most) render them of importance in the origin of tuberculosis, inasmuch as some of the para-tracheal and superior mediastinal glands are only separated by parietal pleura from the lung apex.

“Tuberculosis of the bronchial glands might, in some cases, be derived from the cervical glands, owing to the communication existing between the anterior deep cervical and the broncho-mediastinal glands.

“**Thoracic Glands**.—1. *Anterior or superior mediastinal* glands are situated behind the manubrium sterni, in the angle between the right and left innominate veins, and along the front of the aortic arch. They are close to the thymus gland. These glands receive lymph from the pericardium and thymus gland, and from the internal mammary and tracheal glands.

“2. *Posterior mediastinal* glands are found along the œsophagus and descending thoracic aorta; they receive lymph from the middle and posterior groups of the diaphragmatic glands and from the upper surface of the liver, and communicate with the bronchial nodes; they are therefore a possible source of infection to the roots and bases of the lungs.

"3. *Bronchial Glands*.—These are divisible into four groups:—

"(a) Superior tracheo-bronchial glands, a right and a left set, in the angles between the trachea and the right and left bronchus.

"The right superior tracheo-bronchial glands lie on the right side of the trachea under the mediastinal pleura; they may lead to digestive disturbances by compressing the right vagus nerve. The right group is said to be composed of five to seven glands, the left group of only three to six glands. The latter lie between the aortic arch and the left side of the trachea, and may compress, or injure, the left recurrent laryngeal nerve as it passes upwards in this position.

"(b) Inferior tracheo-bronchial glands, between the two bronchi and in front of the œsophagus and aorta, 9 or 10 in number.

"(c) Broncho-pulmonary glands lie in the angle which each smaller bronchus makes with the main stem of the bronchial tree.

"(d) Pulmonary glands, lymphoid masses situated in the lung and the subpleural tissue.

"The tracheo-bronchial glands derive lymph from the lungs, bronchi, and lower end of the trachea, and from part of the heart and œsophagus. They communicate with the anterior and posterior mediastinal glands, and with the supra-clavicular and tracheal glands of the neck. Their lymph flows to the venous angle between the internal jugular and subclavian veins of each side, and to the supra-clavicular glands; a tubercular infection of the tracheo-bronchial glands might thus lead to infection of the supra-clavicular glands, and of the cervical pleura and lung apex on which the latter glands rest; thus also may be explained the secondary involvement of the supra-clavicular (or lower deep cervical) glands in cancers of the mediastinum and of the lung."

**Symptoms.**—In tuberculosis of the lung or of the tracheo-bronchial glands it is often difficult to make a definite diagnosis, as the whole picture may be that of a somewhat weakly, pale child with capricious appetite and

a physique below that of the normal child of its age. The following, however, are the main symptoms which may be expected:—

Loss of flesh is one of the first points noticed by the parents or guardians of the child. Often it is slight, but at times it is altogether out of proportion to any disease that can be detected in the chest. At other times the statement may be made that the child is not thriving, and that although it has not lost weight there has certainly been no increase for several months. Failure to gain weight over a period of a few months should be reckoned just as important a sign as a direct loss of weight in an adult. When a child is first presented for examination the physician should, invariably, compare its height and weight with those given in the standard anthropometrical tables. In using these tables, however, it must be remembered that they are only averages, and that children vary considerably in their height at the different ages, so that a child who is under the average height should not be expected to be up to the standard weight. In such cases it is better to ignore the age and compare only height and weight. With experience, it is quite possible to form a fair estimate as to the amount of wasting that has been taking place without the aid of the weighing machine, but it is better in all cases, and particularly in all cases of suspect children, to make a careful record of the variations in weight. If a child has failed to put on weight over a few months, then tuberculosis should be discussed as a likely cause.

Loss of strength is another point of importance. As a rule the tuberculous child is listless and easily fatigued. If a child, which has formerly been active and energetic the whole day, begins to hang about in a weary, listless fashion for the greater part of the afternoon and evening, then suspect tuberculosis as a cause. Occasionally these children may have a short lively time in the early evening, but this soon passes off again, and they are more listless than ever. With this there is usually associated pallor of face with a malar flush in the late afternoon or evening.

Cough is a common symptom in childhood, not only of tuberculous diseases of the lung, but of the many other



common diseases to which children, in this climate of ours, are subject in cold wintry weather.

Its importance is thus minimised from a diagnostic point of view. In tuberculosis of the mediastinal glands it may be absent altogether, or be so slight as not to be noticed. If present it may be dry and hacking in character, or it may be paroxysmal like that in whooping-cough. Occasionally it is accompanied by some mucous secretion, but, even though the lungs are seriously affected, the sputum in children is rarely expectorated. Sputum is practically never found in children under five, and rarely, unless the child has been specially taught to spit, in children under fifteen years of age.

Night sweats are an uncertain diagnostic feature, as they are so frequent in non-tuberculous children. They are often a manifestation of rickets, which is such a common disease in the poorer quarters of our cities. Indeed it has been stated that about 50 per cent. of our children show slight rachitic stigmata. Thus, in investigating a complaint of night sweats in childhood, evidence of rickets should be looked for. The time at which the sweating takes place should be carefully ascertained. The night sweats of tuberculosis, as already stated, take place in the early morning, approximately about the hours of 2 and 4; whereas the rachitic sweating, which is confined chiefly to the head and neck, takes place shortly after the patient goes to sleep. Slight night sweating should be reckoned as of little importance from a diagnostic point of view.

Temperature is an important feature in the diagnosis, but it should be remembered that the temperature in the child varies within wider limits than in the adult. A morning rectal temperature of  $99^{\circ}$  F. and an evening temperature up to about  $99.4^{\circ}$  F. might be reckoned as well within normal limits in children up to about fifteen years of age. So, before assuming temperature to be of importance from a pathological point of view, one would require to see it reach  $100^{\circ}$  F., at least, at some period of the day for about a period of a fortnight at least. During that time there might be an occasional day free from fever.

**Inspection.**—As a rule children who are the subject of tuberculosis of the root glands are delicate looking, with poor



physique and imperfectly developed chests. The long narrow thorax is commonly seen. Many of these children have been regarded as likely subjects for the development of tuberculosis, whereas the truth is that they are already suffering from the disease.

Enlarged veins wandering over the front of the chest are common, and if confined to this region alone should raise a strong suspicion in the mind of the examiner. Stoll states that he found them present in 92 out of 173 cases of tuberculosis. Distension of the veins of the neck has been noted in a few cases in children.

Flattening of the upper part of the chest under the clavicles is often found in cases in which the disease has passed on to a cicatricial condition. At times the depression is limited to a round circumscribed area about  $1\frac{1}{2}$  inches in diameter at the inner part of the second interspace, and to this depression the term "hilus dimple" has been applied.

The presence of an overgrowth of soft silky hair in the interscapular region is also noted often in these children. But it is frequently observed in children in whom no other evidence of disease can be found. Another point to be noted in inspection is the presence or absence of slight telangiectasis in the interscapular area. In cases of intrathoracic glandular enlargement there is to be seen at times distension of the small venules in the interscapular area centring around the second dorsal spine. It may be due to pressure from the glands, or more likely to tuberculous thickening of the apical pleura. The sign is not of great importance, as it is seen in a fairly large proportion of children who are apparently quite healthy. Lastly, enlargement of the supra-clavicular glands should be sought for, as, if present, this may be looked on as evidence of some involvement of the apical pleura on that side.

**Percussion.**—In spite of much that has recently been written on the value of percussion as an aid in the diagnosis of tuberculous glands, in my own experience I have found it of very little assistance. Dullness, if present, is found on either side of the manubrium sterni (parasternal dullness). This is frequently found on the left side, but may be attributed rather to the arch of the aorta than to tracheo-bronchial

glands. If a dullness is detected under the manubrium in children up to about six years, the presence of the thymus, as the possible cause of it, should be considered. Posteriorly, there may be found some dullness in the interscapular area, most commonly on the right. To elicit this paravertebral dullness requires an amount of practice in percussion, and, when it is found, it means that the glands are of a very considerable size. When they are small it is not possible to detect them by percussion, and there may be a widespread glandular involvement without any evidence being obtained by percussion. Pain or tenderness is sometimes felt on percussing the thoracic vertebral column in the region of the fourth or fifth dorsal spines, or on pressure over the sterno-clavicular articulations in cases of inflamed root glands, but these are inconstant signs.

**Auscultation.**—The auscultatory signs vary much. At times there may be detected loud bronchial breathing at one or other side of the sternum; at other times it may be heard at one or both sides of the vertebral column in the region of the fourth or fifth dorsal spine. In other cases there are patches of weak breathing heard, or, it may be, the respiratory murmur is weakened over the whole or greater part of the lung. In one case where there was a weakened murmur over the base of one lung, the author found on post-mortem examination a large caseous gland pressing on the main bronchus.

In a normal child, if the stethoscope be placed over the lower cervical spines, well-marked pectoriloquy may be heard. This ceases, with more or less abruptness, over the upper dorsal spines. D'Espine was the first to draw attention to the fact that in enlarged tracheo-bronchial glands pectoriloquy can be elicited for a distance into the dorsal area of the spinal column, varying from the first to the fifth dorsal spine and this has been termed D'Espine's sign. Considerable importance is attached to it in France and America, particularly in the former country, where it is reckoned the most important single sign in the diagnosis of enlarged thoracic glands. To be of any value the pectoriloquy must be well marked. In noting this sign the varying position of the primary bronchi should be remembered. In infancy the

trachea divides about the level of the first dorsal vertebra and by the time the child reaches twelve the division is opposite the fourth.

In estimating the value of this sign the author has to confess to a longing to see the test confirmed on the cadaver to a greater extent than it has been, and he has grave doubts as to its value.

Another sign sometimes used is that of Eustace Smith, which is elicited as follows:—In a suspected case of thoracic glandular involvement get the child to tilt the head backwards as far as possible so that the face is directed towards the ceiling, and if enlarged glands are present then a venous hum may be heard over the manubrium sterni. The explanation of the hum is that it is due to the glands being pressed forward against the superior vena cava or the innominate vein. The test is not a reliable one, and as a hum is present at times in apparently normal children, too much importance need not be attached to it.

**Tuberculin Tests.**—The older the child the less is the value of any tuberculin test. In very young children under one year a positive skin reaction is held to be synonymous with active disease. As the child grows older the value of the test rapidly diminishes. After five years a negative result only is of much value, and then it should be negative on at least two occasions before being accepted. It should be remembered that a negative result is obtained after some infectious diseases, as measles and whooping-cough, and in late stages of tuberculosis. Of the various tests, the von Pirquet cutaneous reaction is the most convenient to apply.

**X-ray Examination.**—Screening is of little assistance in the diagnosis of tracheo-bronchial glands, but much help may be got from a photographic plate. Even this latter, however, is often not of the slightest assistance, as, in many cases, when the physical signs, etc., point towards gland tuberculosis the plate shows no evidence of it. Calcareous glands, of course, show up well, but glands in that condition are not the seat of active mischief. Glands in which there is active tuberculous disease may be producing considerable systemic disturbance, and yet not be opaque enough to reveal their presence in the photographic plate. X-ray

examination here, as in adult tuberculosis, should be looked on as an adjunct to the general examination of the patient.

**Diagnosis.**—The diagnosis of tuberculosis in the root-glands in children is even more difficult than the diagnosis of early tuberculosis in the adult. Ofttimes at the best it is but an intelligent surmise. The general symptoms are common to not a few diseases of childhood. The author has seen the same child diagnosed by a specialist in diseases of children as suffering from chronic intestinal intoxication, and by a tuberculosis expert as being tuberculous. Experiences like this make for a little less dogmatism in any given case, and should teach the necessity for not viewing the child from a small circumscribed point of view. A wide general survey should always be taken. If the constitutional symptoms are well marked, and some localising signs be found, then of course the diagnosis is clear. If, however, no evidence on physical examination of enlarged glands is found, or the constitutional symptoms are ill-defined, then the diagnosis can only be arrived at by a process of exclusion. "When no other cause can be found think of tuberculosis." This is as valuable a dictum as that taught long ago to students when enteric fever was more common than it is now: "In cases of continuous fever think of two diseases—the first should be enteric, the second tuberculosis."

**Prognosis.**—If we are always right in the diagnosis of root tuberculosis in childhood, then the children as a rule make excellent recoveries from it, and tuberculosis of the root glands is a comparatively benign affection. Even in cases where the lung itself is involved, children ultimately do well in the majority of cases. Any physician of experience can point to cases which have, in the end, done well even though the case had reached cavity formation. Nor is the ultimate life of the patient much prejudiced by an attack of gland tuberculosis in childhood. My experience is, that given a reasonable amount of hygienic care no harm comes to the child as he grows up. Like tuberculosis in the adult, the prognosis is influenced greatly by the social and economic position of the child's guardians and by the nurture and care expended on it. The age of the child is also an important factor. The earlier the infection the greater the

risk of fatal complications such as meningitis or generalised tuberculosis.

**Treatment.**—Children suffering from glandular tuberculosis respond, in a striking fashion, to an open-air life. Very often these children come from poor homes in the poorer parts of the town, and may have suffered from insufficient feeding. If such cases can be removed to a sanatorium so much the better, as little else is required. If they must be treated at home then the diet should be supplemented by milk, cod-liver oil, or eggs. General tonics with iron and arsenic are indicated, and a supervision of the child's home by the tuberculosis nurse is desirable.

If the child can get, in addition to its ordinary diet, one to one and a half pints of milk per day, or cod-liver oil, preferably emulsified, up to half an ounce or one ounce of the oil per day, the results are usually most satisfactory.

In order to assist further in this subject it is deemed advisable to introduce the findings of the American National Association for the Study and Prevention of Tuberculosis, on the Diagnosis of Thoracic (Pulmonary, Bronchial Gland, etc.) Tuberculosis in Childhood :—

#### DEFINITIONS.

1. *Loss of Weight.*—By loss of weight should be understood an unexplainable loss of at least five per cent. below normal limits for that particular child, or an unexplainable failure to gain weight over a period of four months.

2. *Loss of Strength.*—By loss of strength, in its pathological sense, is meant ease of tire and lack of staying power which are unusual for that individual child and which cannot be satisfactorily explained.

3. *Fever.*—In young children rectal temperatures alone are dependable. To constitute fever in its pathological sense in childhood there must be a more or less constant elevation of temperature over 100 degrees, taken at various times during the day and lasting over a period of at least one week. In older children temperature rules for adults apply.

4. *Elevation of Pulse.*—No definite standards can be laid down as to what constitutes elevation of pulse, as this varies according to the age. Observation should be over a longer period, and a wider latitude allowed, than in adults, before significance is attached to this as a symptom in childhood.



5. *Hæmorrhage*.—As in adults, any amount of blood, with or without sputum, requires medical investigation as to its source. This is a rare symptom in childhood.

6. *Family History*.—An occasional case of tuberculosis in the patient's uncles, aunts, cousins, etc., should not be considered of importance, unless there has been intimate exposure and personal contact with such a case. It is an important fact, however, when the patient's immediate relatives, as mother, father, brother, sister, nurses, nursemaids, attendants, etc., have been tuberculous, and especially so when there has been prolonged and intimate contact.

7. *Exposure*.—Exposure to tuberculosis, no matter how slight, from human sources, or from milk and milk products, is of very great importance. Prolonged exposure, especially under unhygienic habits or surroundings, is of still greater importance. This question should be gone into with the utmost care.

8. *Cough*.—No cough is characteristic of tuberculosis in childhood. Persistent cough for six weeks requires investigation. Tuberculosis can and often does exist without any cough whatsoever. In certain cases of bronchial gland tuberculosis there may be a brassy, strident, paroxysmal cough resembling that of pertussis.

9. *Sputum*.—Sputum, if present, should be examined. It is comparatively rare in tuberculosis in childhood.

10. *Hoarseness*.—Any huskiness or persistent hoarseness requires investigation. This is likewise rare in childhood.

11. *Râles*.—Râles are not to be regarded as essential in diagnosis and are not in themselves alone evidence of tuberculosis. In fact by the time râles are found in tuberculosis in childhood the disease is usually advanced and the diagnosis only too evident.

12. *Dullness*.—Only very light percussion should be used. Dullness is not to be looked for at the apices, as in adults, but over both sides of the sternum (parasternal dullness), and between the scapulæ (interscapular dullness).

13. *Altered Voice and Breath Sounds*.—Pure bronchial breathing and egophony are comparatively rare in tuberculosis in childhood. Harsh, prolonged, high pitched expiration and an intense whispered voice are often present. The whispered voice and not the spoken voice should be used.

14. *D'Espine's Sign*.—Intense whispered voice heard below the third dorsal vertebra is considered by many as abnormal and indicates the presence of enlarged bronchial glands. Such glands are not necessarily tuberculous however.



## MINIMUM STANDARDS.

On the basis of these definitions the following minimum standards in the diagnosis of active tuberculosis in childhood have been formulated:—

1. Given a definite history of exposure, either from bovine or human sources, any symptoms, constitutional or local, require the most careful investigation, although they may not necessarily be due to tuberculosis.

2. Constitutional signs and symptoms of disease, such as loss of weight and strength, fever, etc., are of more importance than signs and symptoms relating to the chest.

3. Other causes for constitutional signs and symptoms, such as diseased tonsils or adenoids, carious teeth, improper feeding, rickets, etc., should be investigated before these signs and symptoms are attributed to tuberculosis.

4. The presence of tuberculosis elsewhere in the body, such as glands, bones, joints, etc., is not necessarily indicative of tuberculosis in the chest, nor is it true that these forms of tuberculosis convey immunity against pulmonary disease.

5. There may be extensive signs in the lungs, such as dullness, râles, altered voice and breath sounds, without these being due to tuberculosis. On the other hand, active tuberculosis may be present without definite signs and symptoms in the lungs.

6. Tuberculin tests. The von Pirquet skin test is the best to use. When this test, properly applied, has been repeatedly negative on three trials, except during or after an attack of measles, or in the presence of far-advanced tuberculous disease, tuberculosis may be ruled out. A positive skin test in children under five years of age may be indicative of tuberculous disease, and points to the necessity for further observation. The diagnostic value of a positive tuberculin test becomes progressively less important, as significant of active disease rather than infection, in the years from five to fourteen.

7. A definite history of exposure, with a positive tuberculin reaction, accompanied by constitutional signs and symptoms, establishes a diagnosis of tuberculosis, even if the signs in the chest are vague, indefinite, or absent. A similar history of exposure, even with a positive tuberculin reaction, but without constitutional signs and symptoms and without definite signs in the chest, does not justify a diagnosis of tuberculous disease, but merely of tuberculous infection, except in very young children.

8. The X-ray may give valuable confirmatory evidence. A

definite diagnosis of tuberculosis is not justified on X-ray examination alone. In every case the interpretation of the X-ray plate should be made by one qualified in such matters.

9. In all cases in which there is doubt, it is better to make a provisional diagnosis of tuberculosis and to give the child the benefit of hygienic measures and prolonged observation, although this need not necessarily mean that the child be sent to a sanatorium or hospital or be definitely stamped as a consumptive.

10. Finally, a correct diagnosis can be reached only by means of common sense and a careful consideration of a multiplicity of minor signs and symptoms, local and constitutional.

## CHAPTER XVII

### VARIETIES OF TUBERCULOSIS

THE clinical manifestations of pulmonary tuberculosis are very varied, but a few well-defined varieties of the disease are recognised. These are as follows :—

- (A) Acute.—(1) Acute pneumonic tuberculosis ; (2) broncho-pneumonic tuberculosis ; (3) miliary tuberculosis.  
(B) Chronic.—Fibroid phthisis.

(A) **Acute.**—(1) **Acute Pneumonic Tuberculosis.** In these cases the history of the illness approximates that of ordinary lobar pneumonia. There is the sudden onset with rigor and headache, and accompanying it there may be slight vomiting and pain in the chest. Cough, acute fever, and all the classical symptoms of lobar pneumonia are present. Unless care is taken a diagnosis of ordinary pneumonia is made, and it is only when the crisis fails to appear that the physician begins to doubt the correctness of his opinion. A careful examination of the patient, and due consideration of the history prior to the illness, might have enabled the physician to give a more correct diagnosis. In tuberculous pneumonia, for some considerable time previous to the onset of the illness, there is often a period of indifferent health, slight but steady loss of weight, and a trivial but persistent cough. The site of the lesion is often of considerable value, as the apex is the favourite point of attack of tuberculous or as it is sometimes termed caseous pneumonia.

During the course of the illness the temperature as a rule is more fluctuating than in ordinary pneumonia, and there is no terminal crisis. The sputum is less tenacious and more purulent, and in due time contains tubercle bacilli and elastic fibres. During the earlier days of the illness, it

may be streaked with blood, or there may even be definite hæmoptysis. The râles in the chest, in these cases, are of a more bubbling character than in pneumonia, and as the lung breaks down rapidly, the breathing becomes of a hollow bronchial quality. Cavitation may come on within a week. The emaciation is more rapid than in pneumonia, and there is profuse sweating, and profound weakness. Dyspnœa is often an early and marked symptom. Acute pneumonic tuberculosis may terminate fatally in from six to thirteen weeks, but in a fair proportion of these cases, after a cavity has been formed the acute illness subsides, and the disease tends to become of a more ordinary chronic type. If life

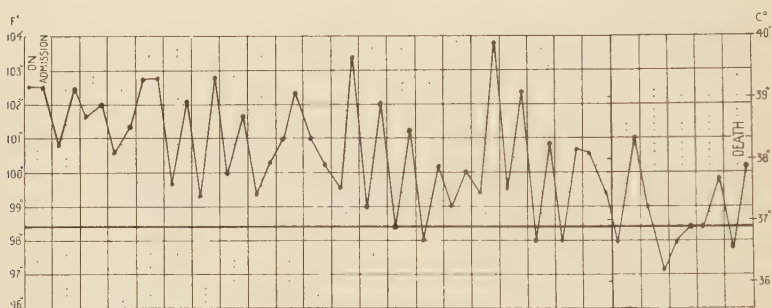


FIG. 39.—Acute Pneumonic Tuberculosis.

is prolonged there is shrinking and limitation of movement on the affected side, with, after a time, some displacement of the internal organs.

(2) **Broncho-Pneumonic Tuberculosis.**—In this variety the history of illness is practically identical with the previous form. The symptoms vary slightly however. The face is more flushed, with often a tinge of cyanosis present; the eyes clear with pearly conjunctivæ. The temperature is higher and with greater fluctuations, and there is profuse sweating. The physical signs are often indefinite. All that may be heard is an occasional crepitation or a few dry rhonchi, scattered about the chest. At this stage a diagnosis of bronchitis is often made, although the temperature, the flushed face, and the sweating ought at once to indicate that the case is not one of ordinary bronchitis. As the disease

advances numerous small centres of consolidation can be noted, which may gradually merge into each other. The respiratory murmur varies. In some spots it is weak and indistinct, and in others it approaches the bronchial type. The lung tends, especially at the apex, to break down rapidly, and then bubbling râles can be noted. The sputum is markedly purulent. These cases almost invariably terminate fatally. Death takes place in from one to two months from the commencement of the disease.

(3) **Acute Miliary Tuberculosis.**—This is often a widespread general infection, but, for clinical purposes, is differentiated into three, according to where the brunt of the attack falls. Thus we have (*a*) meningeal, (*b*) abdominal, and (*c*) pulmonary miliary tuberculosis. We shall deal with the last only.

Acute miliary tuberculosis of the lungs is often but a part of a general infection in which the lung symptoms are the most prominent, and frequently two or more types are present in the same individual. Thus a patient may suffer from a miliary infection of the meninges, and, at the same time, have miliary tuberculosis of the lungs, and the case may be diagnosed as meningitis, because the head symptoms are the more pronounced and attract most attention. In the earlier stages miliary tuberculosis of the lung is often confused with enteric fever, and this is especially apt to be the case if abdominal symptoms are also present. In tuberculosis prostration is more marked and comes on earlier than in enteric, and constipation is more common than diarrhœa. The temperature has not the gradual rise that the enteric temperature has; in tuberculosis it rises more abruptly and fluctuates more. The Widal reaction is of course negative.

In miliary tuberculosis of the lungs the onset is often abrupt and sudden, but just as often there may be a period of ill health previous to the onset with cough, loss of flesh, etc.

In children it may follow whooping-cough or it may be a result of tuberculous infection of some of the lymphatic glands.

The symptoms are varied. Early in the disease cyanosis is a marked feature. There is great prostration and rapid

emaciation. The pulse rate is rapid and the tension low. The temperature is high—it may reach 105° F. and it is irregular and occasionally of the inverse type. The sputum is often mucoid, but at other times it is muco-purulent. In the early stages of the disease, and, indeed, in some cases throughout the whole course of the disease, no bacilli can be detected in it. Hæmoptysis is only occasionally seen.

The physical signs are insignificant and out of all proportion to the constitutional disturbance. They may only be those of a slight bronchitis.

Percussion, as a rule, gives no assistance. Auscultation may reveal a few scattered sibilant rhonchi, but often there are fine scattered crepitations heard over a great part of the lungs. They may not be heard on quiet respiration but may be audible only after coughing. At times pleural friction may be heard in limited areas. As the disease advances the crepitations in the lung become more numerous and coarser; the prostration increases and death takes place often within two or three weeks. The disease practically always ends fatally, although a few cases have been put on record in which it passed into the ordinary chronic type.

The differential diagnosis in miliary tuberculosis of the lung rests on the presence of dyspnoea and cyanosis, the prostration and rapid emaciation and the type of temperature. There is also often the history of a previous attack of some form of tuberculosis.

**(B) Chronic.—Fibroid Phthisis.** This term has been applied to all those cases in which there is a pronounced hypertrophy of the fibrous tissues of the lung. This increase of fibrous tissue may be caused by a variety of diseases. It may be due to an attack of acute pneumonia in which, for some unknown cause, resolution does not take place but the exudate becomes organised into a solid mass of tissue which undergoes a fibroid transformation. This is, at times, spoken of as chronic interstitial pneumonia. It may also be brought about by the inhalation of particles of dust, as in the cases of silicosis or gold-miner's disease. It is also seen as a result of an attack of pleurisy with effusion where the fluid has not become organised. Clinically it is often impossible to distinguish between the various types of fibroid



disease, but it would be well to confine the term fibroid phthisis to those cases where the proliferation of the fibroid tissue is due primarily to the tubercle bacillus. In all cases of chronic tuberculosis there is a production of fibroid tissue, but, in many cases, the ulcerative process proceeds at a quicker rate and results in cavity formation. In the fibroid type fibrosis predominates rather than cavitation. Why an invasion of the tubercle bacilli should produce ulceration at some times, and at others proliferation of the connective tissues, we do not know. It has been suggested that the presence of syphilis makes for fibroid phthisis.

*Symptoms.*—The symptoms of the disease are extremely varied. They depend on the situation and amount of the fibrosis and on the presence or absence of emphysema. This latter complication is of much importance, as when present it colours all the clinical picture, and masks the true underlying condition. When the fibroid changes are confined to one or both apices the symptoms and physical signs are quite different from what they are in cases in which there is massive one-sided fibroid changes. In many of the so-called fibroid apical conditions we are dealing with the cicatricial condition caused by an arrested lesion, but in others there is just sufficient activity in the lesion to produce a slight but gradual extension of the fibroid area.

In these latter cases the patients are usually thin, sallow, tired-looking individuals, with shortness of breath on exertion and small, feeble pulse. Fever is absent, as a rule, and there is little acute systemic disturbance. There is slight chronic cough with scanty mucoid sputum in which no tubercle bacilli may be found, and, occasionally, there may be a slight hæmoptysis. Tachycardia is at times a feature, and palpitation may be complained of. When emphysema is present the sputum is much more abundant and frothy in character. Dyspnœa is more marked and capillary injection of the face and thoracic walls more apparent. The fingers often show pronounced clubbing. The physical signs are chiefly those of emphysema. The chest is barrel-shaped, movement slight; the cardiac and liver dullness encroached on, the ribs more horizontal, and the interspaces wider than normal. The lungs are more voluminous than usual and may cause bulging

above the clavicle on coughing. Inspection may, however, reveal some limitation of movement with some drooping of the shoulder which would point to the underlying tuberculous condition. Myotatic irritability is often present in a marked degree. Vocal fremitus and resonance may be increased on the affected side. The percussion note may be impaired slightly and there may be some disparity on comparison of Krönig's areas in the two apices. Auscultation may reveal nothing except weak breathing all over the chest with some broncho-vesicular or bronchial breathing at the affected apex. At other times the respiratory murmur is harsh with expiration prolonged. As a rule a few rhonchi, or scattered dry crepitations, are heard in the affected areas, and often, also, throughout the lung.

The diagnosis in these cases is often extremely difficult. Numbers of cases diagnosed as bronchitis and emphysema are really cases of fibroid phthisis. The more a practitioner's experience increases, the more cautious he becomes in diagnosing off-hand any case as merely bronchitis with emphysema. At times the diagnosis is further complicated by the patient being stout and well-nourished. X-ray examination is of much importance in forming an opinion, as, if there is much fibroid change in the lung, it shows up very well on the screen. The presence of a dense shadow, however, in one or other apex does not always settle the question, for the shadow may be due to a cicatricial condition which is perfectly healed, but emphysema has supervened and remains the important condition. It is only by prolonged observation and repeated sputum examinations that a final decision can be arrived at.

Where the fibrosis is extensive, or in the later stages of the disease, the diagnosis is easier. There is the marked breathlessness on exertion with cyanosis, the dullness in the chest, the clubbing of the fingers, and usually there are a few bacilli found in the sputum. They may, however, require prolonged searching for before being discovered.

In massive fibroid infiltration of the lung, such as occurs after a pleurisy with effusion, the diagnosis is still more easy. The symptoms are well marked and are almost entirely due to the presence of the dense fibrous tissue in the lung. As

a rule the patient will state that he has never recovered his health properly after the pleurisy. There has always been a slight, irritating cough, a shortness of breath, which has become increasingly worse, and a growing loss of strength. Cyanosis of the face is often pronounced and clubbing of the fingers marked. The sputum varies in quality and character, sometimes scanty, and, at others, abundant and frothy or muco-purulent. As the disease advances, the breathlessness becomes more pronounced, and the patient is incapacitated by anything which demands the slightest exertion.

Physical examination shows marked evidence of retraction. The affected side being smaller than the other, the shoulder may be drooping and the angle of the scapula more prominent. In severe cases there may even be a definite scoliosis. If the left side be affected there is usually some pulsation seen in the second and third interspaces owing to the uncovering of the heart surface by the retraction of the lung. The heart itself is usually displaced. It is dragged over towards the affected side. In some cases almost complete dextrocardia may be produced, and the cardiac impulse may be found under the right nipple, and again in others it may be seen in the left axilla.

The percussion note is, in typical cases, absolutely dull, and there is a feeling of increased resistance to the finger. Vocal fremitus is absent. The auscultatory signs vary considerably. There may be an almost total absence of breath sounds in some cases. In others there may be areas of bronchial or even amphoric breathing. Adventitious sounds may be absent, but as a rule there may be heard pleural friction, rhonchi, a few crepitations, or an indeterminate creaking sound. In X-ray examination there is an extremely dense shadow on the affected side with the ribs crowded together and sloping unduly downwards. The dome of the diaphragm on the affected side is pulled upwards and is much more limited in its movement than the normal, and not infrequently is almost or altogether immobile.

*Course of Disease.*—As the disease advances the shrinking of the fibrous tissue may produce dilatation of the bronchi, thus causing a bronchiectatic condition, and all the symptoms of this condition supervene. In the terminal stages of the

disease the signs of cardiac dilatation are noted. Dyspnœa is profound and œdema of the extremities, with albuminuria, is present. In these massive fibroid cases the unaffected lung as a rule becomes emphysematous, and the anterior border may be found bulging across the middle line towards the affected side. The course of the disease is prolonged over many years, and there can be no doubt but that many of the deaths occurring in the later years of life, which are notified as bronchitis with heart failure, are really cases of fibroid phthisis.

Although the outlook of these fibroid cases is, on the whole, favourable, still there are certain accidents and complications to which they are liable, and which may bring about a fatal issue:—

(1) Continual dragging by the fibrous tissue may produce aneurismal dilatation of one or more blood-vessels, and this tends to produce periodic attacks of hæmoptysis. Should the aneurism be of some considerable size, the hæmorrhage may be so abundant as to kill the patient by suffocation.

(2) If one lung is in a fibroid condition there is always the danger that the other lung may become affected, and, while it is not unlikely that the disease will be of a chronic type, there is always the possibility that it may run a much more rapid course.

(3) If the sputum is copious and purulent for a prolonged period there is a risk of amyloid disease making its appearance in the kidney, spleen, or liver. It is in the fibroid cases of tuberculosis that the laity find comfort when referring to patients with whom they are acquainted, who have lived several decades after having been pronounced as tuberculous or as “having only one lung.” And, indeed, many such patients live useful and active, although somewhat restricted lives, dying in the end of some other intercurrent disease.

## CHAPTER XVIII

### CLASSIFICATION

PULMONARY tuberculosis is a disease of such varied manifestations that, to a casual observer, there would appear to be little in common between many of its phases. This is well seen in comparing one patient who has a slight attack of this disease with another suffering from an acute extensive process in the lungs. In the one case the patient may appear to be in good health, well nourished, and fit for work; in the other there may be pronounced emaciation and such exhaustion that the patient is unable to leave his bed. Because of these wide differences, attempts have been made to group the patients into different categories or stages, and thus it is that a form of classification has gradually grown up.

**Turban-Gerhardt Classification.**—On the Continent, and to a considerable extent in our own land, the Turban-Gerhardt classification has been employed. This classification was adopted by the International Conference on Tuberculosis at Vienna. It is based almost entirely on pathological anatomy. Three stages are recognised, as follows:—

*Stage I.*—Disease of slight severity, limited to small areas, which, for example, when affecting the apices bilaterally, does not extend beyond the spine of the scapula and the clavicle, or when unilateral does not extend below the second rib anteriorly.

*Stage II.*—Disease of slight severity, more extensive than Stage I., affecting, at most, an entire lobe; or of greater severity, extending, at most, over half a lobe.

*Stage III.*—Disease of greater extent than Stage II., and all cases with considerable cavities.



The obvious drawback to such a classification is that it takes no note of the general systemic disturbance. This, after all, is the most important point in connection with the disease. A patient with a small active lesion which might be classed as Stage I. may be in an infinitely worse position, both as regards his present condition and his future expectation of life, than a patient suffering from a quiescent lesion with a large cavity. In such a case, where the outlook for the Stage III. patient is much better than that for one in the earlier stage, it shows that such a classification is manifestly not ideal.

**American Classification.**—In an attempt to improve on this classification, after considerable deliberation the American Sanatorium Association in 1916 adopted the following:—

#### LESIONS.

*Incipient.* Slight infiltration in the apex of one or both lungs, or a small part of one lobe. No tuberculous complications.

*Moderately Advanced.* Marked infiltration, more extensive than under "Incipient," with little or no evidence of cavity formation. No serious tuberculous complications.

*Far Advanced.* Extensive localised infiltration or consolidation in one or more lobes; or disseminated areas of cavity formation; or serious tuberculous complications.

#### SYMPTOMS.

*A* (Slight or None). Slight or no constitutional symptoms, including particularly gastric or intestinal disturbance, or rapid loss of weight; slight or no elevation of temperature or acceleration of pulse at any time during twenty-four hours.

Expectoration usually small in amount or absent. Tubercle bacilli may be present or absent.

*B* (Moderate). No marked impairment of function, either local or constitutional.

*C* (Severe). Marked impairment of function, local and constitutional.

This scheme offers definite arrangements, making feasible the exact labelling of any individual case.

The following combinations are possible:—

Incipient, A.	Moderately advanced, A.	Far advanced, A.
"    B.	"    "    B.	"    "    B.
"    C.	"    "    C.	"    "    C.

For instance "Incipient A" means a patient with an incipient lesion and with symptoms characteristic of the incipient stage, as defined above.



"Far Advanced A," however, means a patient with a far-advanced lesion, but with only incipient symptoms—a combination not infrequently met with.

The various terms employed are at first a little confusing, but this is soon cleared up when it is remembered that Stage I., Incipient, and Early are all synonymous.

Stage II., Moderately Advanced, and Intermediate are also synonymous.

Stage III., Far Advanced, and Advanced are often used to mean the same thing.

For the official records in the City of Edinburgh a somewhat similar classification is used, but substituting the terms I., II., and III. for Incipient, etc., and we have the convenient classification of—

I. A.	II. A.	III. A.
I. B.	II. B.	III. B.
I. C.	II. C.	III. C.

There have been other classifications brought forward from time to time, and one which may be noted is Philip's. This is somewhat similar to the above. He introduces the letter L to signify the local lesion, and the letter S to denote the systemic disturbance. When this is marked a large S is used, and when it is slight a small s.

It is thus possible to have L<sub>1</sub>, L<sub>2</sub>, L<sub>3</sub> stages, and each further modified by the addition of a small or large letter S.

"The presence of complications is indicated by the symbol + followed by a reference to the lesion—*e.g.* L<sub>3</sub>S + ent. tub., indicates a case of extensive lung involvement and vomica formation along with extreme systemic intoxication and intestinal tuberculosis."

One of the most recent classifications is that adopted by the Society of Medical Superintendents of Sanatoria of Great Britain, which is given below :—

Divide (1) all cases by age into—children (both sexes), under 15 years; adults—15 years and upwards; (2) adults by sex into male and female; (3) all cases into pulmonary and non-pulmonary. If pulmonary, a division is made into two classes :—

*Negative Class.*—Cases in which tubercle bacilli have never

been demonstrated at some time in the sputum—the distinction to be based on medical evidence (personal or documentary).

Subdivide each of these two classes into nine sub-groups according to the factors (*a*) anatomical extent and severity of the lesion, (*b*) degree of systemic effect.

(*a*) Anatomical extent and severity of the lesion to be divided into three stadia according to a modified Turban-Gerhardt method.

*Stadium I.*—Lesion of slight severity affecting at most the apices of both lungs not lower than the spine of the scapula and clavicle on each side, or the apex of one lung not lower than the second rib in front and the spine of the scapula behind.

*Stadium II.*—Lesion of slight severity more extensive than Stadium I., but affecting at most the volume of one lobe, or severe disease extending at most to the volume of one half lobe.

*Stadium III.*—Lesion of slight severity more extensive than the volume of one lobe; severe lesion more extensive than the volume of one half lobe.

By lesion of slight severity is to be understood disseminated foci of infiltration or slight fibrosis; by severe lesion, consolidation, excavation, or dense fibrosis: in each case as indicated by the obvious signs. A small area of dry pleurisy should not exclude a case from Stadium I. For the purpose of classification the right upper and middle lobes are to rank as one lobe.

(*b*) Degree of systemic effect, to be divided into three grades:—

*Grade A.*—Constitutional disturbance, absent or slight, as judged mainly by the temperature, pulse rate, and effect on nutrition and strength. For example, temperature after an hour's rest should rarely exceed 99° F. in the mouth at maximum, or 98.2° F. at minimum, or, if higher, should be reducible to the lower figure by a week's rest in bed. Pulse rate after an hour's rest in bed should rarely exceed 90.

*Grade B.*—Intermediate between A and C.

*Grade C.*—Severe constitutional disturbance or deterioration: one or more symptoms present in severe degree; for example, temperature during rest at the maximum persistently over 100.8° F. in the mouth, or 101.3° F. in the rectum; pulse rate during rest persistently over 96. All cases with severe complications, whether tuberculous or not, fall in this grade.

*Note.*—Rectal temperatures are preferable; when mouth temperatures are used, the thermometer should be kept in the closed mouth for at least fifteen minutes. In the case of women add to the

temperature limits given 0.6° F. for the premenstrual rise which may normally occur.

From these three stadia and three grades, nine sub-groups are obtained, from which we can build up the three groups desired by the Ministry of Health :—

Group 1 = I. A, I. B.

Group 2 = II. A, III. A, II. B, III. B, I. C.

Group 3 = II. C, III. C.

The terms used in describing a patient's condition on discharge from a sanatorium are usually as follows :—

- (1) Not improved, *i.e.*, no local or general improvement.
- (2) Improved, *i.e.*, local or general improvement.
- (3) Disease arrested, *i.e.*, no constitutional disturbance, no fever, no tachycardia, slight or no cough and spit. Tubercle bacilli may or may not be present. This condition should have existed for at least three months.
- (4) Apparently cured, *i.e.*, condition as above, but without cough or spit. No tubercle bacilli present in any occasional sputum, and this condition existent for three months. Physical signs those of a healed lesion.

**Charting.**—In chest work it is customary to use graphic symbols to describe the underlying lung condition and to indicate the physical signs. There is an advantage in the method, as it enables one to record, in a rapid and fairly accurate way, conditions which would require a long written description. Up to the present no national mode of charting has been adopted; each worker uses his own, or his own modification of some existing scheme.

The following are those used by the author, and are based, to some extent, on Wylie's :—

## SIGNS.

		Slight.	Moderate.	Marked.	Absent.
Retraction or Flattening . = R.		I	2	3	
Diminution of Movement . = M.		- I	- 2	- 3	- 4
Increase or Diminution of $\int$ = V.R.		+ I	+ 2	+ 3	
Vocal Resonance.	$\int$ = V.R.	- I	- 2	- 3	- 4
Increase or Diminution of $\int$ = V.F.		+ I	+ 2	+ 3	
Vocal Fremitus.	$\int$ = V.F.	- I	- 2	- 3	- 4

					<i>Percussion.</i>
Dullness . . . . =					Marked
" . . . . =					Moderate
" . . . . =					Slight
Hyper-resonance . . . . =					Hyp.

					<i>Auscultation.</i>
Prolonged Expiration . . . . =					
Harsh or Puerile Breath Sounds . . . . =					
Broncho-Vesicular „ . . . . =					
Bronchial or Tubular „ . . . . =					
Amphoric „ . . . . =					
Weak „ . . . . =					
Cogwheel „ . . . . =					
Absence of „ . . . . =					
Crepitations—	Fine				
	Medium				
	Coarse				
	or Bubbling				

Any other quality to be noted at edge of diagram.

Rhonchi . . . . . =	
Sibilations . . . . . =	
Friction—	Fine
	Coarse

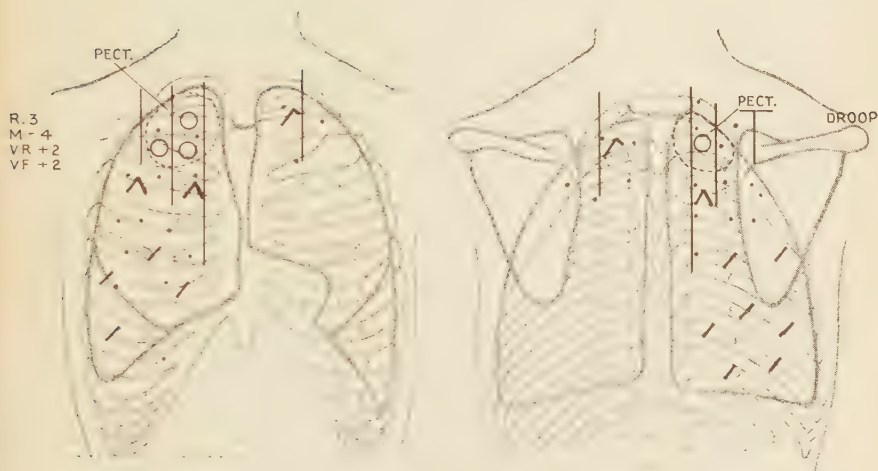


FIG. 40.—Chart of advanced disease, with cavity formation.

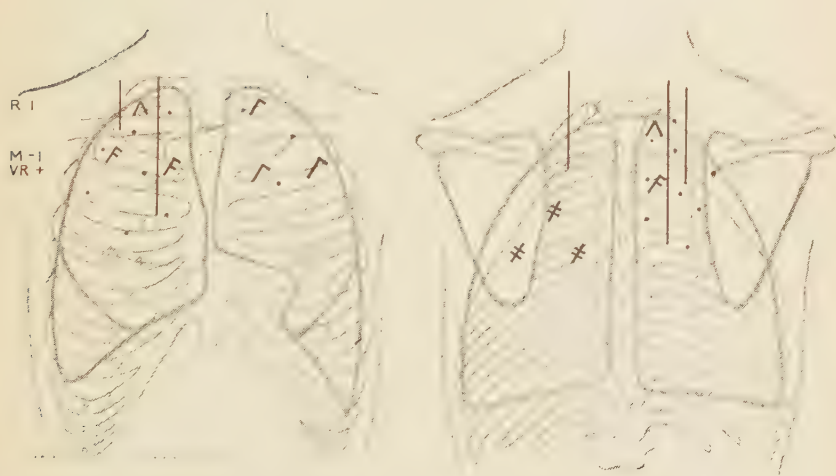


FIG. 41.—Chart of advanced disease, without cavity formation.



## CHAPTER XIX

### DIFFERENTIAL DIAGNOSIS

TUBERCULOSIS is simulated by many other diseases of the respiratory tract, and much attention should be paid to the diagnosis in all cases. There is no difficulty where tubercle bacilli are present in the sputum, but in those cases where it is absent the differential diagnosis is of the utmost importance. To anyone having experience of institutional treatment of this disease it is a well-known fact that a great number of patients are sent to the institutions for treatment who do not suffer from tuberculosis. Unless care is taken, in many instances they are classified as Stage I. of the disease, and they help to swell the number of cures in the Annual Reports of the Institutions. To illustrate the difficulty that exists, it is common knowledge that in the late war many hundreds of soldiers were discharged as suffering from tuberculosis who, at a later date, on careful examination, showed no evidence whatever of any active disease in the lungs.

Many ex-soldiers who have come under my own observation have been so discharged, and, to the best of my belief, did not suffer from the disease at all. Rist, writing in the *Journal of American Medical Science*, instances the fact that 1000 French soldiers had been sent to Base Hospitals from the front line as suffering from tuberculosis, but, after careful observation and examination, he found that of these only 193 were so affected; that is to say, among these soldiers there were over 800 men classified as consumptive who were not so. Again, speaking at the Seventh Annual Conference of the National Association for the Prevention of Tuberculosis, Rist stated, "Thousands of men were discharged on account of a slight suspicion who immediately returned to hard work in the factories, or on the farms, and who never

since developed a symptom or physical sign of tuberculosis." These facts should impress upon us the importance of the utmost care in coming to a conclusion in any given case.

**Disease of the Naso-Pharynx.**—In children who suffer from adenoids and enlarged tonsils, a diagnosis of tuberculosis is often made on insufficient grounds. These children are, as a rule, delicate, pale-faced, slightly undersized, and have a cough with occasional coloured sputum, and, frequently, blood-stains on the pillow in the morning. On examination of the lungs there are frequently found crepitations or rhonchi at either base. These crepitations as a rule are due to the insufflation of muco-pus from the pharynx. They vary from week to week, altering in position, and frequently disappearing. Many of these cases are diagnosed as tuberculous, but, before any such diagnosis is made, the medical man must be satisfied that there are definite apical changes, that is to say persistent crepitations, with slight alteration of percussion note and fever. If the nasal condition is attended to, as a general rule the lung conditions and the constitutional signs disappear. In some of these nasal conditions where there is chronic obstruction of the breathing, some writers have spoken of meeting cases of what they called "induration of the apex." This shows itself by slight retraction at one or other apex, with slight alteration of the respiratory murmur. Cough and spit are usually present, but no tubercle bacilli are ever detected, and there are few definite constitutional symptoms of tuberculosis. The condition, however, appears more likely to be due to arrested tuberculosis.

**Apical Catarrh.**—Occasionally it is found that an apex presents a few crepitations which do not persist more than one or two weeks. This is seen in persons who work at dusty trades, and also in many cases of emphysema. It is quite possible that, at times, this condition may also be due to organisms, other than the tubercle bacillus, such as the pneumococcus or the influenza bacillus. As a general rule the symptoms clear up in the course of a few weeks, and nothing further is heard of these cases. It is quite probable, of course, that some of these are what have been called abortive cases of tuberculosis, but before a diagnosis of

tuberculosis is made constitutional signs of the disease should also be present.

**Chronic Pneumonia.**—In children we frequently meet with basal lesions which have had their origin in an acute attack of pneumonia. The dullness does not clear up, and crepitations persist. As a rule there is cough and spit, but not much systemic disturbance. The child is well nourished, and there is no evidence of fever or night sweats. The only complaint is breathlessness on exertion, the schoolboy being unable to play football as he formerly did: his breathing is not equal to it. These conditions are often supposed to be due to basal tuberculosis. Careful examination of the sputum, however, does not reveal tubercle bacilli, but shows pneumococci, strepto- and staphylo-cocci in abundance. In addition, the general condition of the patient is such as would not be expected in so extensive a lesion if it were of tuberculous character. Emaciation would be severe, night sweats and fever would be present. These cases run a very chronic course and may persist for years. Drug treatment proves to be of little use. Autogenous vaccines usually fail to help the condition. The only therapeutic measure of any value is a prolonged course of treatment on sanatorium lines.

**Bronchitis.**—In acute bronchitis there is always a danger of overlooking an acute tuberculous process, of either the broncho-pneumonic or miliary type of disease. It will help to clear up the difficulties if the following points are noted. In acute tuberculosis the fever is more severe, cyanosis is early and well marked, and sweating more profuse and constant. There is more profound prostration and extremely rapid emaciation. As the disease progresses it will be found that treatment has no effect on the tuberculous condition, and there may arise evidence of a tuberculous invasion of the other organs, such as the meninges, the abdomen, or the choroid. Examination may reveal the presence of old tuberculous lesions in other parts of the body.

In chronic forms of bronchitis there is often a confusion with tuberculosis. In order to help in the diagnosis a careful examination should be made of the chest with a

view to detect any retraction, or dullness on percussion, at the apices; any increase of the vocal fremitus or resonance at either apex, or any alteration in the breath sounds, such as bronchial breathing in the upper parts of the lung, which would indicate some solidification. The localisation, also, of the sounds in the chest is of the utmost importance. Persistent rhonchi, found in the apices of the lungs only, are almost certain to be of tuberculous origin, or if the bronchitis is unilateral it is extremely probable that the cause is tuberculosis. Again and again mistakes are made in such cases and a diagnosis of simple bronchitis given, while the sputum, when examined later, shows tubercle bacilli.

In extensive chronic bronchitis the general health remains comparatively good, and the nourishment practically unimpaired, whereas if the same extensive lesion were due to tuberculosis there would be severe and profound constitutional signs. These bronchitic cases are often complicated by the presence of emphysema, which makes the diagnosis one of extreme difficulty. Here X-ray examination is of considerable help, as this often reveals marked tuberculous involvement of the lung, of which no indication is given in the ordinary examination by percussion and auscultation.

Numerous examinations of the sputum should be undertaken to decide the presence or absence of tubercle bacilli. No case of apparent chronic bronchitis, even with some apical changes, can be diagnosed as suffering from active tuberculosis unless tubercle bacilli are demonstrated in the sputum, for it is quite possible to have a healed tuberculous lesion and bronchitis in one and the same patient.

**Asthma.**—As a general rule there is no difficulty in differentiating asthma from tuberculosis—the attacks being too characteristic, and tuberculosis is not specially apt to supervene in asthmatic subjects. The only point which should be noted is, that asthma beginning in an adult who previously has been free from these spasmodic attacks, should be viewed with the greatest suspicion as being tuberculous in origin, and repeated examinations of the sputum should be undertaken.

**Bronchiectasis.**—This is very frequently mistaken for

tuberculosis, and, on not a few occasions, bronchiectatic conditions in the base of the lung have been demonstrated to students as being due to tuberculous processes. In one case which came under my care a soldier was discharged from the service on the ground that he had a large tuberculous cavity, but, after discharge, he declared quite openly that he knew he had had a bronchiectatic cavity for about a dozen years before enlistment. This shows the necessity for not assuming that all cavitation is tuberculous in origin. In another case, however, under the author's care, a patient had for years been looked upon as suffering from bronchiectasis, but it was found, after his death, that the real cause was tuberculosis.

Bronchiectasis is found frequently in the young, especially as a sequela of measles, whooping-cough, or bronchopneumonia. Other causes may induce it. I remember the case of a young woman who showed signs of what was taken to be a typical bronchiectatic condition, which, she stated, had begun after an operation for a perforating gastric ulcer. Prior to the operation she had been quite well, so far as she knew, but after it she had been seriously ill, had expectorated a considerable quantity of pus, and from that time onwards she was troubled with cough and expectoration. Years afterwards she presented all the symptoms of a bronchiectatic condition.

As a rule in bronchiectasis there is a history of a chronic cough of long duration. The cough is affected, often, by change of posture. Hæmoptysis is not uncommon and a copious purulent sputum is present. The occurrence of hæmoptysis almost invariably is assumed to confirm a diagnosis of tuberculosis. There are certain characteristic points, however, which help in a differential diagnosis. The site of the lesion is almost pathognomonic. Tuberculosis as a rule attacks the apex, and bronchiectasis the base of the lung. Again, in tuberculosis the dullness is confined to one continuous area and has no intervening resonant lung, whereas in bronchiectasis it is not unusual to find areas of dullness alternating with resonant areas. Again, it is not uncommon to find several cavitations present in one lung, while this is infrequent in tuberculosis. In tuberculosis



where the local lesion is marked, we usually find some fever or cachexia, and, it may be, some laryngeal or intestinal complication, whereas in a bronchiectatic condition the constitutional disturbance is often slight compared with the local lesion. There may have been a troublesome cough, with profuse expectoration, for years, and yet the patient be well nourished and show few signs of constitutional disturbance.

The position of the apex-beat often gives valuable information. In tuberculosis the heart tends to be displaced obliquely upwards and towards the side on which the disease is, whereas in bronchiectasis as a rule the displacement is simply lateral. In tuberculosis the apices, as a rule, are retracted, whereas in bronchiectasis, as a result of prolonged cough, we frequently find bulging there. On X-ray examination of the chest tuberculous consolidation is always apparent, whereas the bronchiectatic condition may give few indications of its presence, in spite of marked physical signs. In tuberculosis it is not uncommon to find a low blood pressure, whereas this is not specially affected in the bronchiectatic condition. There are, however, a few cases in which, for a definite diagnosis, reliance can only be placed on the examination of the sputum, and it should be insisted on as an essential in all basal lesions that tubercle bacilli should be repeatedly searched for in the sputum. If, after repeated examinations, no tubercle bacilli are found, even then tuberculosis as a cause of the symptoms cannot be excluded with certainty.

**Empyema.**—In the early stages of this disease there is little difficulty in making a differential diagnosis. There are the usual signs of fluid in the chest, and, as a rule, the dullness is at the base of the lung. A slight confusion may arise in an interlobar empyema, but the onset and course of the disease are so very different from those of tuberculosis that, with care, the diagnosis should be easily made. The greatest difficulty arises in those cases in which the empyema bursts into the lung tissue and the pus is discharged through a bronchus. In such cases there is usually a prolonged period of ill-health, with cough and purulent expectoration, and all the constitutional signs of tuberculosis—emaciation,



sweatings, fever, and occasional hæmoptysis. In the lung there are the usual signs of solidification, or even cavitation. It is at this stage that much confusion is apt to arise. A careful inquiry into the mode of onset of the illness, coupled with the site of the lesion, which is generally basal, and a careful investigation into the sputum, will usually clear up the situation.

**Abscess.**—This may occur in two forms:—

(1) *Acute abscess*, such as is due to foreign body, septic embolism, or aspiration pneumonia. Here there is much systemic disturbance with high fever of intermittent type, with copious purulent sputum, often brownish in colour, and containing elastic fibres but no tubercle bacilli. The site is, as a rule, in the lower lobe of the lung. The differential diagnosis rests on the history, the mode of onset, and on the profound constitutional disturbance, the character of the sputum, and the localisation of the lesion.

(2) *Chronic abscess* is often a result of the acute form. There is a well-marked cavity with dense pus-producing walls. Such an abscess cavity simulates that produced in chronic tuberculosis, and the differential diagnosis is often extremely difficult. A decision will be arrived at by the history of the illness, the situation of the lesion, and by frequent examination of the sputum for tubercle bacilli. X-ray examination often gives valuable assistance, revealing, as it does, the exact situation and appearance of the lesion.

**Gangrene.**—The distinguishing feature about gangrene is the characteristic penetrating fœtor. Unfortunately a tuberculous cavity may become gangrenous, and so the two conditions may be present at the same time, but this is extremely rare. In gangrene the same causes operate as in the causation of abscess of the lung.

The symptoms vary according to the activity of the gangrenous process. As a rule there is marked prostration with heavy sweatings, and high temperature with wide fluctuation. The odour is characteristic and the sputum profuse. When the expectoration is collected for twenty-four hours in a vessel it separates into the well-known three layers—the uppermost, frothy with some muco-purulent matter; the middle, not unlike turbid saliva; and the lowest, thick and purulent,

containing masses of foetid-smelling material made up of pus cells, broken-down lung tissue, blood cells, bacteria, etc.: the whole deposit often greenish or brownish in colour. The differential diagnosis rests on the history, the foetor, the constitutional disturbance, and the expectoration.

**Syphilis.**—This is an extremely rare pulmonary condition, and when it does occur it is usually in the gummatous form. In a somewhat extensive acquaintance with lung conditions, only one undoubted case of syphilis of the lung has come under my observation. It was diagnosed at first as tuberculous infiltration of the apex of the right lung, but, later, the solidification increased and produced some definite bulging of the chest wall with involvement of the sternal ends of the first and second ribs. All the initial symptoms were those of tuberculosis. Under anti-syphilitic treatment all the physical signs disappeared, and several years later there was marked shrinking of the apex with all the classical signs of cavitation. The history of syphilis was quite clear, the Wassermann reaction was positive, and there were at one time gummata on the parietal bones which cleared up under appropriate treatment. The diagnosis of syphilitic lung is really one of trial by ordeal, that is, if anti-syphilitic measures cure the condition, then it is one of syphilis; if they do not cure, then it is probably not syphilis. A positive Wassermann reaction is of little use by itself, as it is quite possible that a true pulmonary tuberculosis may be present, and, at the same time, the patient may have syphilis but not syphilis of the lung.

**Neoplasm.**—New growths in the lung are of two types—(1) non-malignant and (2) malignant. The non-malignant tumours are so extremely rare that they need not here be discussed. The malignant growths are sarcoma and carcinoma.

Primary sarcoma is extremely rare and is usually secondary, spreading from the surrounding parts, or due to metastasis.

Primary carcinoma is more common, but secondary cancer of the lung is the most common form of neoplasm. Both, however, are rare. In a dispensary and sanatorium practice, dealing with just over 1000 new cases of chest disease per

annum, only about 12 cases of neoplasm were seen in the past seven years, and they had nearly all been previously diagnosed as tuberculosis.

In primary cancer of the lung the symptoms at an early stage are often vague, and simulate those of pulmonary tuberculosis. There may be cough, slight wasting, dyspnœa, and fever, so that the differential diagnosis is often a matter of extreme difficulty. In some of the cases just referred to, the diagnosis of tumour was easy, but in others it was more difficult. Among the chief points to be attended to, in coming to a diagnosis, are the following:—Dyspnœa is often an early and prominent symptom, and this is not a feature of tuberculosis. Indeed, it is surprising to what an extent the destruction of the lung by tubercle can be carried without any dyspnœa. In neoplasm the paroxysmal character of the dyspnœa forms an important link in the diagnostic chain. Pain is common in neoplasm, in the affected side or down the arm, but pain is rare in tuberculosis in the absence of pleurisy. A sense of oppression in the chest is often found in tumour, and to ease this, as well as the pain and dyspnœa, the patient will often adopt a seemingly awkward position in bed when he finds that such a position gives relief. This is not found in tuberculosis. Cough is not a diagnostic feature in tumour, and, indeed, in early stages it may be absent. Fever may be present in both conditions, so is of little help in making a diagnosis. Cachexia is usually pronounced in the later stages of tumour, the complexion being of a yellowish colour, and the skin parchment-like. The cachexia is easily distinguished from the appearance in late tuberculosis.

*Physical Signs.*—The unilateral character of neoplasm is characteristic. It is extremely rare to find both sides affected, whereas in late tubercle it is exceptional to find the disease confined to one lung. The situation and character of the dullness caused by tumour is distinctive. It is unusual to find the apex affected. The rule is that the dullness appears in the middle area of the chest, and the upper part of the lung gives a clear note, whereas in tuberculosis the dullness is generally at the apex, and diminishes as we descend. When the tumour reaches the periphery of the lung and involves the pleura, the percussion note becomes of a

particularly stony character. No other condition gives quite the same note. It is just as if one were percussing a block of marble.

The breath sounds are, at times, of assistance. In neoplasm there will often be an area of marked dullness, but instead of finding, as in tuberculosis, bronchial breathing and crepitations, it may be noted that the respiratory murmur is weakened or altogether absent, and no crepitations are heard. Alterations in the outline of the chest and displacement of the heart are often marked, and give valuable aid. In tumour, in late stages, the side affected is frequently enlarged, and the heart is displaced by being pushed over towards the healthy side, whereas in tuberculosis there is retraction of the affected side, and the heart, if displaced, is drawn towards the affected area. X-ray examination is of considerable assistance. In neoplasm the dark shadow is seen springing from the root of the lung and extending out towards the periphery, whereas in tuberculosis the shadow is most pronounced at the apex, and gradually shades off towards the lower portion of the lung.

Blood in the pleural sac is said to be a point of diagnostic importance in cases of tumour, but, in a majority of cases, it is not met with. Indeed, the only case in which the author has recently met with blood in the pleura was one in which there was not even a suspicion of new growth. Sputum examination must not be neglected, as when expectoration is present it may occasionally furnish valuable evidence. If the tumour invades a bronchus some of the tumour cells may be expectorated, but this is not very common. Pressure signs are often of great importance in cases of neoplasm. The venous return, being restricted by pressure on the large venous trunk within the chest, there is marked distension of the veins in the neck or upper part of the chest—this condition being absent in lung tuberculosis. All the above detailed points are not to be found in any one case, though several of them may be present, and it is only by carefully noting, and estimating the value of, those present that a diagnosis is arrived at.

**Actinomycosis.**—This is a rare disease which simulates pulmonary tuberculosis closely. There are the usual

signs of cough, expectoration, emaciation, and fever. In the later stage the sputum is copious and there may be hæmoptysis. The site of the actinomycosis is usually the middle or lower lobe of the lung, but occasionally it may be found in the apex. The diagnosis is made, usually, by demonstrating the fungus in the sputum. Occasionally the disease spreads to the chest wall and forms a fluctuating tumour, which when opened contains pus with the characteristic growth.

**Streptothricosis of the Lung.**—This is another rare disease of the lung which simulates tuberculosis. There are the usual signs and symptoms of chronic phthisis, but there is stated to be less emaciation. The writer has been on the look-out for a case of streptothricosis for several years, but has so far failed to find one, so apparently rare is it.

**Hydatid of the Lung.**—This is very uncommon in our country, but cases have occurred. The symptoms are usually cough, spit, shortness of breath, and hæmoptysis.

The differential diagnosis is difficult, and it is only when the cyst has ruptured into a bronchus and particles of the membrane of the cyst, or the hooklets, are coughed up that the diagnosis can be definitely made. If the case is seen after the cyst has been discharging for some time into a bronchus, the diagnosis is still more difficult as the cavity often becomes septic, and there may be some breaking down of the lung tissue itself, so that the sputum contains elastic fibres, thus further simulating tuberculosis. Tubercle bacilli are, of course, not found in the sputum.

**Hæmorrhagic Bronchitis.**—A few cases of this disease have been reported in recent years. The symptoms closely resemble those of tuberculosis, the distinguishing feature being the discovery in the sputum of the spirochæta bronchiales.

**Pulmonary Infarction.**—This is occasionally met with in mitral disease, and because of the fact of the associated hæmoptysis, it is at times confused with tuberculosis. The symptoms vary according to the size and number of the infarcts. There is usually slight sudden dyspnœa, pain in the chest with cough and hæmoptysis. This last varies in amount but is rarely more than a few ounces, and comes on



a few hours after the onset of the attack. The physical signs vary. Slight dullness may be detected, but, more frequently, only a faint pleuritic rub with a few moist sounds are heard. With care and attention to the history of the illness and the condition of the heart, a correct diagnosis should be easily arrived at.

**Mitral Stenosis.**—The results of this condition are often mistaken for pulmonary tuberculosis. There are cough, expectoration, wasting, blood-spitting, and often some fever. The chest may show some apical crepitations, the result of previous infarctions. There may be numerous râles in other parts of the chest. Not a few such cases have been sent into sanatoria as cases of tubercle. In all cases of suspected tuberculosis the heart should be carefully examined, and when a mitral murmur is detected, particularly one due to stenosis, it should be an axiom that tuberculosis must not be diagnosed too readily in the absence of tubercle bacilli from the sputum.

**Hyperthyroidism.**—Recently attention has been drawn to the close similarity between the group of symptoms associated with slight degrees of hyperthyroidism and those of early tuberculosis. There is the languor and "easily-tired" feeling of early lung disease, tachycardia, sweating on the least exertion, and often slight fever. Careful examination of the chest, however, shows no evidence of disease. As a means of making a sure differential diagnosis, American observers have recommended that a small subcutaneous dose of adrenalin be administered. If the case is one of hyperthyroidism, the tachycardia will be at once made worse after administration. In order to carry out the test, the patient should be in bed for some time prior to the injection and the pulse rate carefully recorded.

## CHAPTER XX

### COMPLICATIONS

THE tuberculous complications met in dealing with cases of pulmonary tuberculosis are extremely varied. This is well illustrated by the following table, which is taken from the admirable treatise on *Diseases of the Lungs*, by Hartley and Powell. From autopsies on 263 consecutive cases of chronic pulmonary tuberculosis the undermentioned details were obtained:—

COMPLICATIONS					
	Males. Per cent.	Females. Per cent.		Males. Per cent.	Females. Per cent.
Larynx. . .	54·7	46·8	Kidneys—		
Trachea . .	27	20	Ulcerative Tuber-		
Main Bronchi .	12·2	10·6	culosis . .	2·6	1·3
Pneumothorax .	7·4	4·0	Bladder, Tuber-		
Pleurisy, serous .	6·3	...	culosis of . .	1	...
„ suppurative . .	2·1	2·6	Suprarenals, Tuber-		
Aneurism . .	10·6	5·3	culosis of . .	2·1	2·6
Fatal Hæmoptysis	9·0	4·0	Male Generative		
Pericarditis . .	2	...	Organs . .	4·7	...
Thrombosis of			Tuberculous Dis-		
Veins . .	1·0	6·6	ease—Testicle	1	...
Peritonitis . .	3·7	2·6	Epididymis . .	3	...
Tuberculous ul-			Vesicles . .	2	...
ceration of			Prostate . .	3	...
Intestines .	61·3	61·3	Female Genera-		
Fistula. . .	1·6	...	tive Organs . .	...	10·7
Lardaceous Dis-			Tuberculosis—		
ease . .	9·5	13·3	Ovaries . .	...	2·7
Kidneys—			Tubes . .	...	10·7
Miliary Tuber-			Endometrium .	...	6·7
culosis . .	11·1	9·3	Meningeal Tuber-		
			culosis . .	2·6	2·6

**Tuberculosis of Pleura.**—Primary tuberculosis of the pleura is an extremely rare event, but secondary infection of this membrane is a common occurrence in the course of acute or chronic tuberculosis of the lung. In studying pleurisy where it occurs as an apparently initial illness we are at once met with the question whether it is a manifestation of an already existing pulmonary tuberculosis or not. There is no doubt that some cases of pleurisy are not of tuberculous origin. They may be the result of a rheumatic infection, or may be caused by pneumococcal, streptococcal, or staphylococcal invasion. Pleurisy also occurs as a complication of mediastinal neoplasm; and pleural effusion is not uncommon as a terminal manifestation in cardiac or renal diseases. It may also be due to traumatism.

The fact that many patients have had acute pleurisy with effusion, and have never shown any clinical evidence of tuberculosis at a later date, must not, however, be accepted as full evidence of its non-tuberculous character. While it is freely granted that pleurisy is, at times, of a non-tuberculous origin, in the majority of instances, especially where associated with effusion, it is due to the tubercle bacillus. The evidence of this is twofold, viz. (*a*) clinical, and (*b*) bacteriological.

(*a*) *Clinical Evidence.*—When the after-history of patients who have had pleurisy is followed, it is found that a considerable number ultimately develop tuberculosis of the lung. We meet with this regularly in patients who date their lung trouble from the time when they had pleurisy. Bowditch's figures are worthy of quotation. Following up 90 cases of pleurisy which had been attended by his father or himself between the years 1849 and 1879, he found that 32 had either died, or were suffering, from tuberculosis. Numerous other observations have been made on the point, and it is generally conceded that from 30 per cent. to 40 per cent. of patients who suffer from acute pleurisy ultimately develop phthisis.

(*b*) *Bacteriological Evidence.*—An ordinary microscopic examination of the pleural exudate rarely shows tubercle bacilli. This is most probably due to the fact that their presence is difficult to demonstrate owing to the paucity of their number and the amount of fluid present. The suggestion has also been made that the effusion may possess

some bacteriolytic power and thus cause a disappearance of the bacilli. Whatever the reason, they are not found in more than 1 or 2 per cent. of the cases.

If, however, special methods are adopted in investigating the effusion, the bacilli can often be demonstrated.

Jousset, by his method, which he terms Inoscopy, states he was able to demonstrate the bacilli in 17 successive cases. He adopts the following procedure:—

The fluid is drawn off from the chest, allowed to clot, and digested with artificial gastric juice. It is then centrifugalised and the sediment stained in the ordinary way by the Ziehl-Neelsen stain.

By inoculation experiments it is often possible to demonstrate the tuberculous character of the effusion. One observer injecting 15 c.c. of the exudate into the peritoneal cavity of a guinea-pig found that tuberculosis developed in 65 per cent. of the cases. Another worker, by injecting large quantities, up to 50 c.c. or over, demonstrated its tuberculous character in 85 per cent. of his cases.

Thus, clinically and experimentally, it is proved that in cases of pleurisy with effusion the majority are probably due to the tubercle bacillus. Recognition of this fact should ensure that more care be taken of the patient during an attack of so-called idiopathic pleurisy, and for a prolonged period afterwards he should be subjected to careful medical examination at stated periods.

The situation of the inflammatory patch may be at the apex, in the interlobular region, on the convex surface of the lung, or on the diaphragmatic surface. The site is in great part determined by the situation and extent of the primary lung disease, being found usually in the pleura overlying a tuberculous focus.

**Symptoms and Signs.**—The symptoms vary according to the position of the inflamed area, and according to whether the pleurisy is or is not accompanied by effusion. Apical pleurisy is extremely common; indeed, it often is the first symptom which calls attention to the underlying lung disease. There is pain in the shoulder or in the subscapular region. In some cases dilatation of the pupil on the affected side is seen, and, occasionally, enlargement of the supraclavicular

glands. Auscultation may reveal a definite friction sound or some crepitation-like sounds in the supra-spinous or supra-clavicular areas. The respiratory murmur is usually weakened. There may be some of the slight constitutional symptoms which are associated with early phthisis.

In interlobular pleurisy there is usually no friction to be detected, but the patient complains of pain and is feverish, and has an irritating cough. There may be no evidence of disease on physical examination, but after repeated attacks there is usually some dullness over the site, with diminution of the respiratory murmur and evidence of pleural thickening and retraction. X-ray examination furnishes valuable diagnostic aid, as it shows a dense shadow in the region of the interlobular fissure.

In diaphragmatic pleurisy there is often marked pain which is not necessarily confined to the skin surface nearest the inflamed point. In many cases it may be referred to the abdomen and simulate appendicitis, peritonitis, or cholecystitis. As a rule there is tenderness on pressure along the diaphragmatic attachment posteriorly. The true nature of the abdominal pain may be realised by noting that deep pressure over the area complained of does not cause an increase of pain, as it would do were there an underlying inflammatory process. In some other cases the pain may be complained of in the neck, along the edge of the trapezius.

The physical examination will show diminution of movement on the affected side, and X-ray examination will reveal the fact that the diaphragm is almost immobile. In dry pleurisy in the lateral region of the chest, which is the more common situation, there are the usual signs of pain, more or less intense, slight irritating cough without sputum, and immobility of the chest. There may be some hyperæsthesia of the skin over the affected area. Auscultation may reveal typical friction with diminished breath sounds due to the deficiency of movement. X-ray examination in a recent slight case may reveal nothing abnormal. As a rule there is a moderate degree of fever with a varying amount of constitutional disturbance.

**Pleural Effusion.**—When effusion takes place there is usually a moderate amount of fever varying up to 102° F.



and 103° F., although occasionally it may apparently be absent or so slight as not to attract attention. The fever when present usually lasts three or four weeks, and then gradually subsides. Breathlessness is often marked, although this depends on the amount of effusion.

*Physical Signs—Inspection.*—When the effusion is copious the side affected appears larger than the other, and there is lessened respiratory movement. In extreme cases it may be immobile. The apex beat, if visible, is displaced towards the healthy side. Cyanosis and dyspnoea may be observed at times.

*Palpation.*—The interspaces are wider than usual and the ribs more horizontal. Vocal fremitus is usually absent, and this is of great assistance in outlining the edge of the effusion. If the finger-tip be used, the difference between the fremitus just above, and just below, the margin of the fluid is sufficiently well marked to be of valuable assistance in delineating the margin. Occasionally a definite thrill can be detected on tapping the chest wall.

*Percussion.*—The percussion note is absolutely dull. The dullness is often accompanied by an ill-defined thrill which is detected by the finger. This thrill, if present, distinguishes the dullness of an effusion from that produced by lung tumour or by densely thickened pleura. The dullness diminishes towards the upper part of the fluid, but with care, and by using delicate percussion strokes, the margin may be defined with tolerable accuracy. The upper border is not horizontal but is curved in S-shape fashion, being higher in the axillary region and lower anteriorly and posteriorly. This has been associated with the names of various observers. It has been termed Ellis's, Garland's, or Damoiseau's curve.

In extreme cases of effusion, by careful percussion the edge can be detected bulging over the middle line and encroaching on the other lung to a variable extent. This is usually seen in the region of the third or fourth rib. The extent of the displacement of the heart should be carefully mapped out by percussion. This is often difficult, but the information which it gives renders the attempt desirable. In uncomplicated cases there is always displacement of the heart, but when there are old adhesions this may be interfered

with, and there may be a large effusion and yet no apparent cardiac displacement. Above the fluid level the lung often gives a curiously qualified percussion note. It has a tympanitic quality added, and is termed "Skodaic Resonance" after the writer Skoda who first drew attention to it. This peculiar resonance is not always present, and is stated to be due to the relaxed condition of the lung above the fluid level.

In the unaffected lung, percussion will sometimes reveal a triangular area of dullness, situated close to the vertebral margin, which forms one boundary. The apex of this dull area is at the level of the fluid on the other side, and the external border is a line drawn from the apex to the base of the lung at a point two or three inches from the vertebral column. This area has been termed Grocco's paravertebral area of dullness. The sign is not of much importance, nor indeed is it always present. The signs of pleural effusion are identical whether the effusion be serous, hæmorrhagic, or purulent. Attempts have been made to distinguish them by physical signs, but the only sure method is by paracentesis.

*Auscultation.*—As a rule the breath sounds are much diminished if not entirely absent. Vocal resonance is also much interfered with. This lessening of the resonance may also be made use of in outlining the upper border of the fluid. If the stethoscope be moved carefully up and down the chest wall in the neighbourhood of the edge of the fluid, the alteration in the intensity of the sound may be sufficient to enable the examiner to distinguish when he is over fluid and when over lung. At times the conduction of the whispered voice sounds are of more assistance than the actual voice sounds.

The diminution or complete loss of the breath sounds does not necessarily follow in effusion. There are times, and this is especially frequent in children, when the breathing is purely bronchial in character and even pectoriloquy may be clearly elicited and yet the chest may be full of fluid. In children it is often impossible without paracentesis to decide definitely whether dullness is due to a fluid effusion or a solid lung.

The diminution or absence of the breath sounds in cases

of effusion is usually explained in the following way. The vibrations in the lung, when they meet the pleural surface and pass from that into another medium, namely the fluid, lose force, and in the fluid these diminished vibrations are wellnigh lost and are unable to penetrate the chest wall. Ægophony is well marked. It is of no special diagnostic significance, although Laennec thought it was pathognomonic of fluid. It is heard, at times, in pure solidification of the lung.

**Diagnosis.**—The diagnosis of pleural effusion, when the lungs are fairly normal, is usually comparatively easy. The large immobile side, the displaced heart, the dull percussion note, and the absence of vocal fremitus and breath sounds make an easily recognisable picture. When the effusion takes place with an already diseased lung, or in a case where there are already extensive adhesions, the diagnosis is more difficult and, often, can only be definitely settled by paracentesis. The heart, instead of being displaced away from the effusion, may already be displaced towards it because of the contraction of pre-existing fibrous bands. On the other hand, the displacement may be away from the effusion for the same reason. Thus we may have the anomaly of a left-sided effusion with the apex-beat external to the left nipple line, or, vice versa, a right-sided effusion with the heart drawn to the right.

*X-Ray Examination.* — X-ray examination may give valuable assistance. In small, dry pleurisy of recent origin, no alteration is detected, but when the pleura is thickened a dull grey patch will be found which does not light up on inspiration. After repeated attacks it is not always possible to settle definitely whether it is the lung or the pleura which is involved. In interlobar pleurisy there may be seen, when the effusion is moderate in amount, a dark band running transversely across the lung in the situation of the interlobar division. This band is of one density throughout, as distinguished from tuberculous deposits which vary in density. At a later stage the band may become more oval or circular in shape, owing to the pressure of the effusion between the lobes. When the effusion is absorbed there is sometimes left a thickening, or sclerosis, of the pleura in that

region, and this may show, under proper illumination, as a dark, narrow band running across the lung in the region of the interlobar division.

In diaphragmatic pleurisy with effusion, either serous or purulent, there may be seen a dark shadow running along the top of the diaphragm, the outline of which is obscured. At times it may be difficult to state whether the shadow is above or below the diaphragm. In a large general pleural effusion there may be complete opacity. The upper surface of the diaphragm is indistinguishable, and the movement altogether abolished. The displacement of the heart and of the central median shadow is readily seen. In children it is often difficult to observe the effusion, and the displacement of the heart is less than in adults.

After the effusion has become absorbed it is sometimes difficult to note any appreciable difference on the two sides, but usually there is a lack of illumination in the affected side and some interference with the movement of the diaphragm. In small effusions there is loss of illumination in the costo-diaphragmatic sinus and some limitation of the excursion of the diaphragm on the side affected. X-rays give no assistance in distinguishing the nature of the effusion. This can only be settled, with any degree of certainty, by paracentesis.

**Treatment.**—In the earlier stages of pleural effusion little or no active treatment is necessary, beyond rest in bed. No undue haste should be shown in drawing off the fluid, as by placing the affected lung at rest, the fluid may be having a most beneficial effect on the underlying tuberculous condition. After three weeks or so, or if there be signs of pressure, evidenced by cyanosis or dyspnœa, paracentesis should be performed and the fluid withdrawn. The amount may vary from one to three or more pints, and, in the cases where dyspnœa has been a marked feature, relief will be obtained at once. In non-tuberculous cases of effusion, the fluid, as a rule, slowly subsides and recovery takes place—the lung slowly expanding as the fluid is absorbed. This can be noted by the return of the breath sounds to places where formerly they were absent. Friction sounds may again reappear, as the fluid is absorbed. In many cases of undoubted tuberculous effusions the fluid will permanently

disappear in a few weeks, but in others it will recur, and remain for an indefinite period, and paracentesis thoracis may be necessary.

*Paracentesis Thoracis.*—The site usually recommended for puncture is in the mid-axillary line in the sixth interspace, but some prefer the posterior aspect just below the angle of the scapula. The advantages of these sites are: (1) that they are well out of the neighbourhood of the heart or diaphragm, so that there is little danger of either of these organs being injured; (2) the position is convenient when the patient is reclining in bed, and it is clear of the mamma; (3) the parietal wall is thinnest in this region.

The fluid may be syphoned off or aspirated, but whichever method is employed strict aseptic precautions should be taken.

It is rather a shock to discover, after finding a serous effusion in a chest on the first operation, that, on a later occasion when the fluid is again removed, it has become purulent. The conscientious practitioner may wonder if any fault lies at his door owing to a neglect of surgical cleanliness. Effusions which recur again and again do often in the end become purulent, but the operator should certainly not contribute to this by his lack of aseptic measures.

The patient is placed near the edge of the bed in a comfortable reclining position with the arm on the affected side raised and thrown over to the opposite side, so as to increase the space between the ribs. The skin should be purified, and it may be frozen by ethylchloride spray. If the fluid is to be syphoned off, all that is necessary is a trochar and cannula, with a piece of rubber tubing attached, about three feet or so in length. The end of the tube can be placed in some antiseptic solution in a receiver. This is all that is requisite in the majority of cases. Some operators prefer to use Potain's aspirator (see Fig. 42).

When all is ready for the operation the skin at the selected point should be pulled down slightly, and the space between the ribs carefully noted by pressing the point of the index finger of the left hand deeply between them. The needle is then firmly and steadily passed in over the upper edge of the lower rib. The skin may, with advantage, in



nervous cases be anæsthetised by subcutaneous injection of a 3 per cent. solution of eucaine, or by freezing. When the needle is withdrawn the skin should be pinched firmly between the thumb and index finger, and a small piece of cotton-wool and collodion placed over it. It is then permitted to resume its normal position, and because of the slight preliminary dragging down of the skin, the puncture wound is more effectually sealed.

In purulent effusion there is no difference in the physical signs in the chest, but there is a marked difference in the

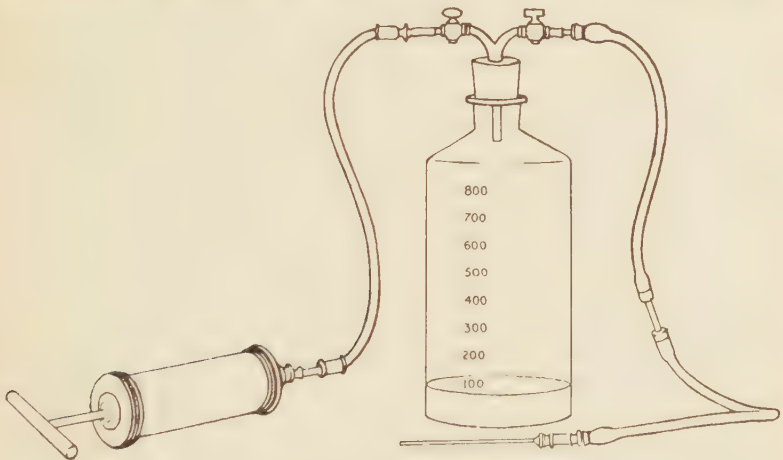


FIG. 42.—Potain's Aspirator.

prognosis. The treatment is unsatisfactory. If the abscess is converted into an open cavity by operation, the lining membrane continues to secrete pus, and the lung often fails to expand completely. We are then left with a chronic sinus in the chest wall leading to a large abscess cavity, which becomes infected with secondary organisms and continues indefinitely to pour out pus. The patient drags out a miserable existence of a few months, or at most a year or two, and, in the end, succumbs to amyloid disease. The results of major operations of the Estlander type are not always satisfactory, although a few such are remarkably successful.

**Tuberculosis of Larynx.**—This is a complication which

often adds distressing features to a case of lung disease. It is more common than is often suspected. In a series of over 1000 cases under my care, it was present in 30 per cent. It is more common in men than in women, and is rare in children. The severity of this complication varies greatly. It ranges from a slight infiltration to marked ulceration and œdema of the larynx.

Laryngeal tuberculosis is always secondary to the lung disease. When ulceration is present, it appears to mask the lung signs, as, in many cases, there is a great disparity between the apparent lung condition and that of the patient generally. The patient may be obviously going down day by day, and yet, with the exception of the throat condition, there are few signs of tuberculous disease to be detected. This may be accounted for by the loss of function at the glottis interfering with the proper production of the respiratory sounds in the chest. Whatever the explanation, laryngeal ulceration masks the physical signs in the lung.

**Symptoms.**—*Hoarseness* is an early symptom. It varies in degree. The voice may have merely lost its clearness of tone, or the patient may only be able, with much apparent exertion, to produce a hoarse, rasping sound. Slight hoarseness should always lead to a careful examination of the larynx. It may be due to a simple catarrhal condition, caused by the passage of irritating sputum, or to definite, though slight, tuberculous deposit. It must not be assumed, although a definite tuberculous lesion is not detected, that the condition is necessarily one of simple irritative laryngitis, for the tuberculous deposit may be so minute as to be incapable of detection by the naked eye. If the lesion is non-tuberculous it usually passes off with treatment, whereas the tuberculous laryngitis is extremely intractable.

*Aphonia.*—In some cases the hoarseness passes on to complete aphonia owing to the lesion being so situated that it has caused a thickening or erosion of the free edge of the cord, or such a degree of swelling that its movement is interfered with.

In a few cases the aphonia is due to paralysis of the cord, which may be brought about by pressure on the recurrent

laryngeal nerve either by thickening of the apical pleura or by enlarged cervical glands.

*Pain.*—This is a really distressing feature, and unfortunately in some cases the pain is almost continuous. Every inspiration appears to accentuate it, and no picture is so pathetic as that of the aphonic consumptive attempting to tell of his continuous laryngeal pain. It may at times be caused only by swallowing food, and then the patient adds to the seriousness of his condition by taking a minimum of nourishment. The pain may be confined to the larynx, or it may radiate up towards the ear or side of the head on the side affected. In late stages of the disease diagnosis of this complication is easy, but in the early stages it is difficult. When ulceration has set in, the only difficulty, and it is a real one, is to distinguish the tuberculous from a syphilitic condition. In the latter case the ulcers are often more sharply punched out, but the clinical difference is not often very decided. There are, of course, always the history, and the Wassermann reaction, to be relied on in forming an opinion.

It is often, also, difficult to distinguish between a simple parenchymatous thickening on the posterior wall, between the arytenoids, and a tuberculous deposit; but in the former case there is no surrounding inflamed area.

The tuberculous lesion may begin in various regions. If it is confined to one cord, this will show redness, and, it may be, some local swelling, while the other cord remains normal. This unilateral affection is always in favour of tuberculosis. Again, it may begin in the commissure, the processus vocalis, the arytenoids, or the wall of the larynx itself.

The laryngeal appearances vary widely: there may only be slight thickening of the cord, with or without erosion. From this, all conditions are met with up to total destruction of the cord, with partial or total destruction of the epiglottis. Pallor of the larynx, which has been looked on as evidence of tuberculosis, is but a sign of a general anæmic condition. Sometimes the epiglottis or the arytenoids are so cedematous that a view of the interior of the larynx is impossible.

**Prognosis.**—Tuberculous laryngitis adds to the gravity of the outlook—the worse the throat condition, the worse the prognosis. Patients with pronounced laryngeal ulceration rarely recover, but in some cases with extensive superficial erosion recovery takes place under suitable treatment. There is no doubt that many of the so-called simple laryngitis cases in consumptives who recover are really tuberculous conditions.

**Treatment.**—There is no preventive treatment for laryngeal tuberculosis. When the disease is established the best line of treatment is to ensure complete rest for the larynx, and to secure this the patient should be forbidden to speak, communicating all his wants and wishes in writing. This is extremely irksome and trying to some patients, but in institutions it is frequently accomplished and kept up for months with great success. If this rigorous treatment cannot be carried out in its entirety, then the prohibition of the use of the spoken voice is the next best, the patient being encouraged to converse in a quiet whisper.

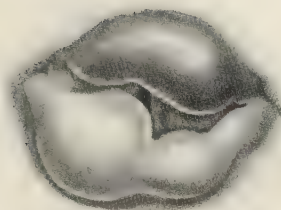
Where there is actual ulceration of the larynx, it is well to spray it frequently with an alkaline solution such as the following:—Sod. chlor., gr. v; sod. bicarb., gr. x—to one ounce of water, warming it to an agreeable temperature. Painting the ulcerated surface with lactic acid was formerly much used, and beneficial results have been observed from such a practice. The usual practice was to begin with a 1 per cent. solution and increase slowly, as the larynx was able to tolerate it, up to a strength of about 10 per cent.

Where there is pain this may be relieved by injecting into the larynx a 10 per cent. solution of menthol in parolene. If pain is severe a spray of cocaine solution, 5 per cent. strength, will alleviate the distress. In those cases where swallowing is mainly responsible for the pain, the cocaine spray is best. Orthoform is another excellent anodyne, and it may be given by insufflation.

Still another means of relieving pain is by injecting 2 to 3 c.c. of a solution containing 80 per cent. alcohol and 1 per cent. cocaine into the neighbourhood of the superior laryngeal nerves. It is not necessary to inject into the



(a)



(b)



(c)

FIG. 43.—(a) Laryngeal tuberculosis with granuloma on the right vocal cord, with an inter-arytenoid ulcer. (b) Acute laryngeal tuberculosis, with considerable oedema of the epiglottis and the arytenoids. (c) Tuberculosis of the larynx. Portion of the epiglottic cartilage has been destroyed, and there is a large granuloma springing from the inter-arytenoid membrane.





substance of the nerve itself. The relief from pain often lasts for some weeks after the injection. Tracheotomy is occasionally performed in an attempt to alleviate the throat condition, but seldom with any success.

**Abdominal Tuberculosis.**—This may be considered with advantage under two headings: (1) intestinal tuberculosis, and (2) tuberculous peritonitis.

(1) **Intestinal Tuberculosis.**—Different observers state they have found this complication in post-mortem examinations, in from 60 to 80 per cent. of cases dying from pulmonary tuberculosis. There is definite ulceration of the intestinal mucosa of varying depth. Some ulcers may reach the peritoneal coat and ultimately rupture. Usually the ulcers are multiple and affect the Peyer's patches and solitary glands. They have characteristically thin undermined edges with an irregular infiltrated base. Some tend to spread transversely but others are nearly circular. They show little tendency to heal, but when they do, may produce stenosis of the bowel.

*Symptoms.*—In the ulcerative variety the prominent symptoms are colicky pains in the abdomen—the frequent passage of loose motions, often dark in colour from the presence of minute quantities of blood, and with a fœtid smell. This diarrhœa is of extremely intractable character. Occasionally, however, constipation alternates with the diarrhœa. Pressure over the abdomen may elicit pain especially in the ileo-cæcal region. Blood is frequently found in the stools in minute traces as “occult blood”—at other times there may be definite and repeated hæmorrhages.

*Treatment.*—When there is chronic diarrhœa in a tuberculous patient one is apt to think at once of tuberculous ulceration, but it is not always due to this cause. This diarrhœa may be caused by the swallowing of sputum, by the tuberculous toxin, by unsuitable food, or by ulceration. It is often extremely difficult to decide between these causes. In children the question of rickets has also to be considered. Where the diarrhœa is due to ulceration the patient should be placed in bed and kept there. His diet should be light and easily digested, and such as will leave little

residue in the intestine. For the relief of pain, hot applications may be applied to the abdomen. To check the diarrhoea any of the numerous astringents may be used, but as a rule opium in some form or other is ultimately called for.

(2) **Tuberculous Peritonitis.**—From a clinical point of view tuberculous peritonitis may be divided into two main groups: (1) ascitic, and (2) adhesive.

*Ascitic Peritonitis.*—In this form the noteworthy symptom is the prominent abdomen, due to the effusion of fluid. It might be noted here that fluid in the abdomen usually escapes observation until there is approximately 1500 c.c., and this is only detected by percussing in dependent parts of the abdomen, or in the knee and chest position. There is usually abdominal pain, a varying amount of fever, occasionally vomiting, and often considerable emaciation which is due in great part to the lung disease.

*Adhesive Peritonitis.*—This form may be local or widespread. In extreme cases the peritoneal cavity may be totally obliterated, and the coils of bowel so matted together as to form a tumour-like swelling. In other cases the omentum is thickened and puckered up, giving rise to a transverse sausage-like tumour, and there may be some encapsuled ascites. As a rule there is pain, but at times this condition gives rise to no pain. There may be felt, occasionally, by careful palpation, some abdominal friction. Diarrhoea and obstinate constipation may alternate, and even complete stoppage of the bowel may occur.

The results of the treatment of tuberculous peritonitis vary. Many of the ascitic cases recover completely under proper open-air conditions, combined with good feeding. Mild counter-irritation to the abdomen, as by iodine ointment, is often beneficial, and improvement may also occur by the inunction of mercurial ointment, or on the administration of cod-liver oil and syrup of iodide of iron. Operative interference in the form of a simple laparotomy often achieves wonderful success in the ascitic variety, but equally good results have accrued from tapping with trochar and cannula. The adhesive form is particularly intractable to treatment, and this is altogether symptomatic.

**Genito-Urinary Tuberculosis.**—During the course of pulmonary tuberculosis, secondary infection of various parts of the genito-urinary tract not infrequently occur.

**Tuberculosis of Kidney.**—Albuminuria is not uncommonly present in advanced tuberculosis, but this does not necessarily mean that there is a tuberculous infection of the kidney. Post-mortem results show that definite disease of that organ is present in more than 30 per cent. of the patients who die of the pulmonary form of the disease. In an early stage there is no pain or discomfort, but as the disease progresses there is usually lumbar pain which may only be elicited on deep palpation. There may be pain referred to the bladder or even to the thigh on the affected side. The urine is usually abundant, of low specific gravity, contains albumen, and sometimes hyaline casts. As the disease advances, pus or blood will frequently be detected in it.

*Treatment.*—This is extremely unsatisfactory. Medicines have apparently little or no effect. General hygienic and dietetic treatment should be carefully attended to, and if the kidney symptoms are so pronounced as to eclipse those of the primary lung focus, the question of nephrectomy may require to be discussed.

**Tuberculosis of Bladder.**—This is not a common complication but it does occur. The symptoms are those of an ordinary cystitis, *i.e.* pain, frequency of micturition, and the passage of urine containing blood and pus. The treatment is unsatisfactory, and in the main symptomatic. Irrigation of the bladder by antiseptics is stated to have been beneficial in some cases.

**Tuberculosis of Testicles and Seminal Vesicles.**—A secondary involvement of the testicle in lung disease is not uncommon. The disease usually begins in the epididymis, and may spread to the body of the testicle. It may also pass upward, along the vas deferens, or involve the skin of the scrotum. The disease is at first painless, and may be discovered by accident. There is a definite swelling felt behind the testicle, which as it increases produces a dragging sensation. At a later stage the skin over the swelling may be involved and a tuberculous abscess form which bursts and leaves an indolent sinus.

Treatment, if not surgical, consists in securing the general improvement of the patient's health and in the support of the testicle by a suspensory bandage. Excision of the testicle may be performed with advantage if done early, but many patients prefer to permit the local disease to continue its own natural course rather than submit to the operation. Tuberculosis of the seminal vesicles is not a common complication; when present it may manifest itself by frequent and painful micturition. Palpation per rectum will reveal the enlarged vesicles.

**Tuberculosis of the Female Genital Organs.**—This occurs occasionally in the course of lung disease. The lesion begins as a rule in the Fallopian tubes, and spreads to the contiguous organs. The symptoms are those of a chronic inflammatory condition of the pelvic organs. Surgical interference is probably the best line of treatment.

**Tuberculous Meningitis.**—This occurs occasionally during the course of the primary lung disease, its onset being gradual and very insidious. The symptoms vary greatly, with headache usually as an initial sign. Indeed, a persistent headache, even without other signs, in the course of a case of consumption, should be looked on with the gravest suspicion. In addition to the headache, vomiting is usual and is often accompanied by constipation. The vomiting may be unaccompanied with nausea and be without any apparent cause, and this is of diagnostic value. As the disease progresses, evidence of irritation to some of the special nerves is seen, in the form of facial paralysis, optic neuritis, photophobia; Kernig's symptom is usually present at an early date; memory is affected, the patient becomes irritable when questioned, and shows signs of confusion of ideas. The pulse is usually slow and the temperature elevated. As the later stages of the disease approach, there may be drowsiness, involuntary micturition, convulsions, coma, and death.

Lumbar puncture may assist in the diagnosis. Occasionally in the fluid drawn off the tubercle bacillus can be demonstrated. The cytology of the fluid is not of much value, as in many cases it shows no change, while in others lymphocytes abound.



**Pneumothorax.**

This is a grave and often particularly distressing complication in the course of pulmonary tuberculosis. It was formerly thought to be wellnigh always fatal, but this is not so. Cases are met with which appear to make a complete recovery.

**Cause.**—As a rule pneumothorax is secondary to pulmonary tuberculosis, but, occasionally, it occurs in what appears to be a normal individual. On one occasion an engine-driver, who had never had any previous illness, was suddenly seized while at work with pain in the side and acute breathlessness. At an examination later he presented the classical signs of complete pneumothorax, and in due course made an excellent recovery. In such a case it is problematical whether tuberculosis was the cause or not. Of course it is quite possible to have a small local tuberculous focus situated under the pleura, so small as to escape detection, and this may rupture and cause pneumothorax. Healing may take place during the time the lung is at rest, and no further trouble arise. Another explanation of such cases is that there may have been pleural adhesions which, on some sudden exertion by the patient, caused a tear of the visceral pleura, thus permitting an escape of air into the pleural sac. In other cases emphysematous bullæ may form around an old healed tuberculous nodule, and their thin walls rupturing, may give rise to pneumothorax. In the majority of cases pneumothorax takes place in recognised cases of lung disease.

**Symptoms.**—In a classical case of pneumothorax there is usually sharp, acute, and sometimes agonising pain in the side affected. There is pronounced dyspnœa and evidence of shock. The pulse is small and rapid. There is cyanosis of the face, frequent shallow respirations, or even orthopnœa. Death may occur suddenly before the initial shock has passed off. While these are the common symptoms, sudden, acute, alarming, and even fatal, there are a few cases in which they are all absent, and the pneumothorax is discovered accidentally. Occasionally, in a patient suffering from pulmonary tuberculosis who is confined to bed, a routine

examination of his chest will reveal a complete pneumothorax, and the patient may be entirely ignorant of anything abnormal having happened. There is no pain, no distress, and no symptoms of any kind, to indicate what has taken place.

**Physical Signs.**—The signs of pneumothorax are very distinctive. The affected side is larger than the healthy one, the interspaces are wider, the ribs more horizontal. The shoulder is elevated, and the whole side is immobile. This immobility contrasts strongly with the exaggerated movements of the healthy side. The apex-beat may not be visible, but if it is, it will be seen to be displaced towards the affected side. Vocal fremitus is absent. The percussion note varies according to the tension of the air in the pleural cavity. Where there is no great pressure the note is hyper-resonant, or even tympanitic, but where the pressure is high the note may be dull. The difference in tension of the air is due to the character of the pleural perforation. If the opening be such as to permit free passage of air in and out of the pleural cavity, then the pressure will be that of the atmosphere—that is, it will not be increased; but if the perforation be valvular and so arranged that it permits the entry of air to, but prevents its exit from, the cavity, then the tension may be raised to ten, twenty, or even more centimetres of water. When hyper-resonance is present it will be noted that the area oftentimes extends over the sternum into the other side for a varying distance. The respiratory murmur is, as a rule, completely absent over the greater part of the chest. In some cases, however, there can be heard faint, but characteristic, cavernous breathing. At the apex there is often heard a weak respiratory murmur owing to the fixation of the lung there by pleural adhesion. The bell sound can be elicited, as a rule, by tapping a coin placed on the chest wall with another coin. Metallic tinkling is often heard to perfection, and the voice sounds frequently have a well-marked amphoric quality.

Later, when effusion into the pleural sac has taken place, and a condition of hydro-pneumothorax is produced, other classical signs result. There is a dull area at the base of

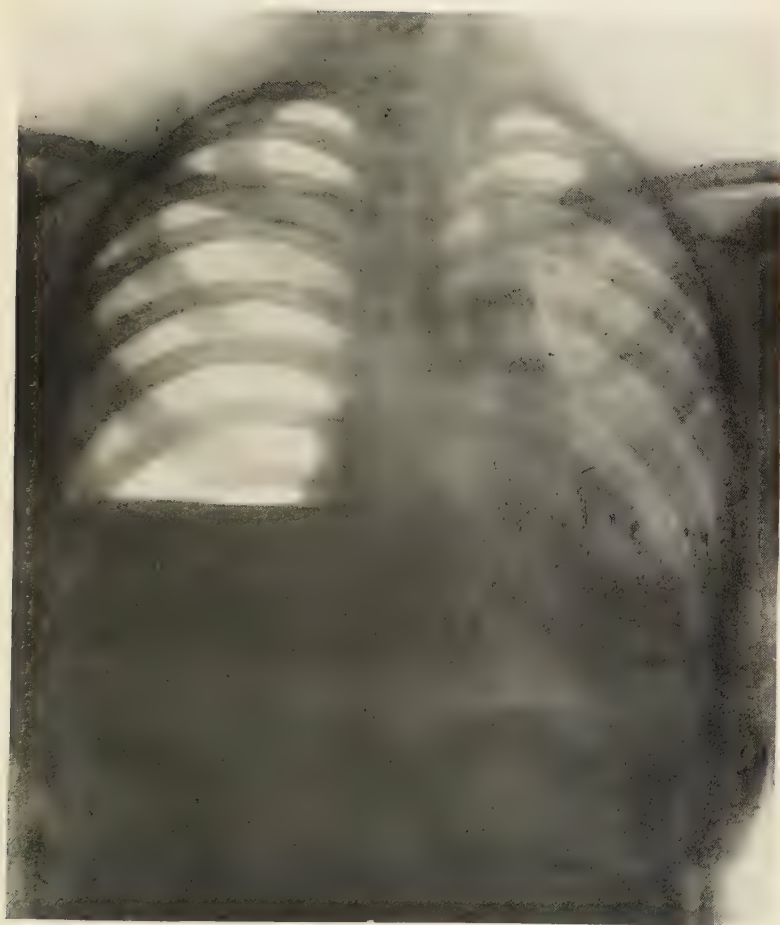


FIG. 44.—Pyo-pneumothorax. Antero-posterior position.



the lung, but this area can be modified by altering the position of the patient. This alteration of fluid level is beautifully seen on X-ray examination. The upper edge of the fluid is at one level and clearly shown, in contradistinction to the upper margin of the fluid in an ordinary pleural effusion. The surface may be seen in motion owing to the pulsation of the heart being communicated to the fluid. When, however, the patient alters his position from the vertical to a more horizontal one, the surface of the fluid alters correspondingly, and this is pathognomic. In a case of complete collapse of the lung, the pleural cavity is more translucent than the healthy side, and the lung can often just be recognised, pressed flat against the central line.

Another typical sign of hydro-pneumothorax is the splashing sound which is heard when the patient jerks himself about. He himself hears it, and knows how to produce it. In one rare case a patient came for advice because he had noted this peculiar sound in his chest, and this was the first indication that he was the subject of a hydro-pneumothorax.

**Diagnosis.**—The diagnosis of pneumothorax, as a rule, is easy. The large immobile chest, the cardiac displacement, and the hyper-resonance, combined with the symptoms and history, make a picture not easily mistaken. A difficulty arises in cases of partial pneumothorax where the lung is bound down by adhesions and prevented from completely collapsing. In these cases there may be sudden pain and a rapid appearance of the signs of a cavity which would indicate pneumothorax, but, when pain is absent, it is difficult to distinguish a partial pneumothorax from a large phthisical cavity. The dyspnoea of asthma has been mistaken for that of pneumothorax, but a careful examination of the chest will clear up the difficulty, as in asthma there are usually scattered rhonchi throughout both lungs.

**Prognosis.**—As a rule, pneumothorax is a grave prognostic indication. In the majority of cases, the complication takes place in patients already seriously stricken with tuberculous disease in the lungs, so that death often quickly results, although a certain proportion of patients make quite a



good recovery. Where the primary symptoms are severe, the patient may die in a few minutes from the time of onset. In other instances he passes through the initial danger and may live with a collapsed lung for several months. Again, a serous effusion may take place and the hydrothorax persist for an indefinite period. In one such case under my care the lung has been completely collapsed for about two years. The fluid became purulent, and there now appears to be a communication between the pleural cavity and a bronchus.

**Treatment.**—The treatment of pneumothorax is often, in the first instance, the treatment of shock. The severe pain is best treated by administration of morphia subcutaneously. This will also alleviate the dyspnœa. If there are signs of increasing pressure in the chest, as shown by greater displacement of the heart, increased dyspnœa and general distress, then the puncture of the chest by a fine needle will often give instant and permanent relief. An ordinary hypodermic needle is quite satisfactory for this if care is taken to close the end with the finger during inspiration.

Strapping the chest so as to immobilise the side affected is useful in preventing an increase of the air tension, when once it has been diminished. When effusion takes place in the pleural cavity there should be no undue haste to withdraw the fluid. If, however, it gives rise to pressure signs or discomfort, it should be withdrawn and replaced by air. Should the fluid become purulent, and the lung show no sign of expanding, it should be drawn off repeatedly. If in spite of this the patient does not show evidence of improvement, the question of more extensive operation for allowing the chest wall to fall in, should be discussed.

Many other complications are met with in pulmonary tuberculosis, such as thrombosis of the veins of the legs, peripheral neuritis, keratitis, etc., but a description of these is beyond the scope of this work, only the more important having been dealt with. For the others reference must be made to the literature of general medicine, or, as in the case of keratitis, to works on the special diseases of organs.

## CHAPTER XXI

### PROGNOSIS

PROGNOSIS in any case of phthisis is always a matter of difficulty, and frequently little more than guess-work. There are many instances in which an excellent prognosis has been given by clinicians of wide repute in cases which have terminated fatally within a short time of the examination. Contrariwise, any practitioner of experience must have met with individuals in the prime of life, or in a robust old age, whose boast it was that they had been condemned as hopeless cases of consumption in early life. Yet, in spite of the undoubted difficulty of giving an accurate prognosis, the practitioner has often to give an opinion as to the probable course of the disease, and the following points will assist in so doing:—

**Social Status.**—It has been said that no poor man can afford to have tuberculosis, thus indicating that a successful issue is more likely to take place in the well-to-do, rather than in the poverty-stricken or working-class patient. A moment's consideration will show the truth of this. The working-class patient has a constant fight for the essentials of existence. When he falls a prey to the disease, the financial circumstances of his home become still more straitened, and he may suffer from insufficient feeding. If removed to a sanatorium his stay there is almost always far too short to complete a cure. He returns to his poor surroundings and his toil, only in too many cases to succumb ultimately to the disease.

The well-to-do patient, on the contrary, if sent to a sanatorium, can stay as long as is necessary, and when he leaves the institution can make the preservation of his health his first consideration.

**Age.**—In the first two years of life, tubercle of the lungs is very frequently fatal. Few of those attacked recover from it. In girls about the age of puberty there are indications that the prognosis is worse than at earlier or later years. In the aged, especially if associated with emphysematous changes in the lung, or arterio-sclerosis, the progress of the disease is usually slow. Nevertheless this is not always so, and, at times, in elderly people the disease makes rapid progress.

**Heredity.**—A bad family history, as a rule, makes a bad prognosis. Where either of the parents, or brothers, or sisters have died of the disease, a slight attack in a previously healthy member of the family should be looked on as a grave matter. When more than one member of the patient's family has succumbed to tuberculosis, it means that we are dealing with an individual in whom that intangible thing, resistance to tuberculosis, is diminished.

**Mode of Life.**—A condition of lessened resistance to tuberculosis is brought about by various other circumstances. It is found in those who lead a life popularly described as "burning the candle at both ends," "fast livers," etc., persons guilty of excesses, alcoholic, sexual, or otherwise. It is met with in others living a life of toil by day, and of exhausting study by night. Unduly exhausting labour of any sort, especially if associated with insufficient diet and unhygienic surroundings, contributes to it, as do also frequently recurring pregnancies or prolonged lactation. These, and other like factors, increase the susceptibility to the disease, and add corresponding gravity to the prognosis.

**Past Tuberculous Diseases.**—An examination of the patient may reveal evidence of some past tuberculous disease which has been recovered from. This is commonly tuberculous glands in the neck. Experience shows that if tuberculosis attacks the lungs of a person who has already battled successfully with the disease in the cervical glands, his chances of overcoming the lung disease are enhanced thereby. It would appear that a certain degree of immunity is produced by the previous attack. Similarly, if there is evidence of an old healed lesion in the lung, it can be taken as presumptive evidence that the individual is again likely to resist the new invasion.

**Present Tuberculous Complications.** — In contradistinction to healed tuberculosis, present active tuberculous disease of other organs darkens the prognosis. The only exception to this rule is the case of slight non-suppurative disease in the cervical glands. Tuberculous ulceration in the larynx adds gravely to the prognosis. Abdominal tuberculosis, ulceration of the intestine, tuberculous nephritis or extensive joint disease, all, likewise, make the outlook dark. The prognosis is made worse according to the extent, severity, and degree of exhaustion which the intercurrent tuberculous complications have produced in the patient.

**Extent and Nature of the Lesion.** — Generally speaking, the more extensive the lesion the worse the prognosis, and bilateral disease is less hopeful than unilateral. In making these assertions, other factors, such as temperature, etc., come in and modify the outlook, and so have to be taken into account, but these will be dealt with later. Where there is much contraction of the chest wall indicating the prevalence of extensive fibroid changes, the progress of the disease is, as a rule, extremely slow, and a comparative old age may easily be reached by these patients. Evidence of rapid breaking down of lung tissue, as shown by the development of cavities, the presence of elastic fibres in the spit, or the progressive invasion of new areas of lung, can be taken as indicative of a bad future. In miliary tuberculosis the gravest prognosis can be given.

**Constitutional Symptoms.** — Pronounced emaciation and a bad prognosis go together. Fever is probably the best indication we have as to the activity of the disease; the higher or more fluctuating the temperature, the worse the prognosis.

The pulse rate is of much importance; it ranks next in significance to the temperature. A rapid pulse with low tension is indicative of profound toxæmia, and carries with it a correspondingly grave outlook.

An anæmic condition should always be looked upon as an indication of a toxæmia, and the more profound the anæmia, especially if it does not respond to treatment, the worse the prognosis. Persistent anorexia, with or without definite gastric disturbance, adds greatly to the gravity of

the outlook, as can be readily appreciated. Disturbance of sleep, whether due to coughing, sweating, or other causes, retards recovery. The presence of marked night sweats is another indication of toxæmia, and adds a corresponding gravity to the prognosis.

**Hæmoptysis.**—Slight hæmoptysis is of little or no value in forming an opinion as to the ultimate course of the disease, but repeated hæmorrhages are not good. The more often the bleeding takes place, and the greater the quantity lost, the worse is the outlook. A single severe hæmorrhage gives little or no guidance as to the future.

**Tubercle Bacilli in Sputum.**—It has been shown that the chances of recovery are much greater in the cases of tuberculosis where the bacilli are not present in the sputum than in those where it is present. This of course is to be expected, as the presence of the bacilli means a breaking down of the lung tissue.



## CHAPTER XXII

### TREATMENT

**Climatic Treatment.**—In the treatment of tuberculosis much less attention is now paid to change of climate than was formerly done. This possibly may be because we are now called upon to treat a greater proportion of poor people who cannot, for financial reasons, consider climatic treatment. There is, however, another reason, and that is because we now know that there is no climate in the world which will with certainty cure pulmonary tuberculosis, and that equally good results have been obtained in sanatoria situated in very diverse climates.

It is well that the advantages of the various climates should be understood, as, at times, patients desirous of a change to another climate have to be advised. The chief types of climates are (1) marine; (2) inland.

(1) Marine climates have a more equable temperature than those inland. The diurnal and annual variations of temperature are less. The air is moist, free from impurities and micro-organisms. The effect on the patient is that respiration is slower, the heart-beats stronger, and the peripheral circulation increased. The advantages of a marine climate can be secured by sea-voyaging or by residing at the sea-coast.

Ocean voyages were highly recommended by a past generation of medical practitioners, but it is not common to recommend them now. This is not altogether due to a mere whim of fashion, but chiefly to the fact that suitable institutions for the treatment of this disease can be found in all climates, and to a recognition of the truth that the careful medical supervision of a patient is more than climate. Again, the modern passenger ship is about the last place that an invalid

should be in, if he has to spend his nights in a stuffy cabin and his days with a crowd on deck. The sudden changes of temperature incident to modern steam-boat travel are trying. Within a couple of weeks the patient may be hurried from the tropics to the vicissitudes of an English climate. In the days of sailing-ships it was different. A voyage to Australia and back might occupy eighteen months or longer. The voyage was made leisurely, and if the patient was well enough to assimilate his food, often crudely cooked and monotonous, he would likely do well. Before recommending a sea voyage it is well to know the patient's fitness for sailing, particularly with regard to his liability to sea-sickness. This is an important point, for nothing can be more exhausting to a consumptive than a prolonged attack of sea-sickness. Only those in the very early stages of the disease should be sent. Any case with cavity or with the disease in an active condition accompanied by fever, or where there is much emaciation and anorexia, or a tendency to hæmoptysis, should not be sent on a sea voyage.

The marine climate can be enjoyed by residence at the coast, where the majority of the advantages and none of the disadvantages of ocean travel are met with. If care is taken in selecting a place removed from a manufacturing district, purity of air can be assured. The types of cases suited for treatment at the sea-coast are those where a fair amount of emphysema is present or where there are heart or kidney complications. Aged patients with atheromatous vessel changes, or with chronic bronchitis, do best at the coast. Young children with glandular tuberculosis also do well at the seaside.

(2) Inland climates are roughly divisible into three classes: (i) low; (ii) medium; and (iii) high altitudes.

*Low Altitude.*—This includes any altitude from sea-level up to about 1000 feet. The climate depends on the proximity to the sea, whether situated on the east or west side of the country, and on the relation to the hills. All sanatoria in Britain are situated between these levels.

*Medium Altitude.*—This includes any height from 1000 to 3000 feet. There is nothing distinctive about the climate

at these heights. Local factors, as in the low altitudes, play an important part in forming the climate.

*High Altitudes.*—This includes any altitude from 3000 up to about 6000 feet. The characteristic features of the high altitude climate are the purity of the atmosphere, the abundance of sunshine, the dry, cold winters and the lessened barometric pressure. The types of case best suited for treatment at a high altitude are incipient cases, or those with only slight destructive changes in the lung, and few evidences of constitutional disturbance. The contra-indications for high altitudes are cases complicated with emphysema or cardiac disease, cases where there is much destruction of lung tissue with cavitation, fibroid cases with dyspnœa or where there are grave tuberculous complications. The general results of the high altitude are increased activity in respiratory and cardiac functions, improvement in appetite and in general metabolism.

It should be emphasised, however, that tuberculosis can be cured in any climate, and that the patient should be cured, if possible, in the climate in which he expects to live after recovery. Careful medical supervision, attention to the details of treatment, careful alimentation, are all of much more importance in the treatment than climate. Still there is an aspect in the climatic treatment which must be noted, and that is the profound, subtle mental influence which a change secures. A very slight alteration in the patient's environment will produce it. It is not necessary to travel very great distances to secure it, and yet this mental change may be the basal factor in all climatic treatment. The result of this change of environment is well known in the non-tuberculous individuals who are suffering from over-work or from the effects of disease. The over-worked man has lost the joy of labour, and the convalescent cannot think of resuming his task. After a few days amidst surroundings totally different from those of the work-a-day life, the over-worked man recovers his elasticity of body and mind, the convalescent renews his strength, and each looks forward with pleasure to resuming his labours. The greatest effect of the change is mental. This aspect of the treatment of the tuberculous should never be forgotten. A patient may be

sent from home and the stimulus of the new environment will be easily seen in his physical improvement. After a time, however, he ceases to respond, a longing to return home comes over him, and no further beneficial results need then be looked for, but the change on returning home again may produce a further improvement.

**Sanatorium Treatment.**—The “open air” treatment which has been so much practised in sanatoria is not new. In 1840 Dr George Bodington wrote his *Essay on the Treatment and Cure of Pulmonary Tuberculosis*. In this he condemned the methods that were then in vogue in the treatment of the disease, namely, confinement to hot, stuffy, ill-ventilated rooms; bleeding, calomel, etc. He recommended, instead, nutritious food with exercise in pure air, especially cold, dry, frosty air. His pamphlet can still be read with interest and profit. It is needless to say that, like many another prophet, he was cast out by his brethren. By a strange irony the institution in which he first attempted to carry his open-air theories into practice had to be closed and was later reopened as a lunatic asylum.

The seed sowed by Bodington took root, like many other of our ideas, in Germany. In that country Brehmer, who was influenced by Bodington’s essay, started his sanatorium in 1854, and this may be said to be the institution from which all others sprang. In the latter part of the last century there was great activity in the erection of sanatoria, and many injudicious statements were made as to the results they would achieve. Some enthusiasts apparently had the idea that all that was necessary to abolish pulmonary tuberculosis was to erect sanatoria all over the land, and a course of a few months’ treatment in them would cure practically all the existing cases of the disease.

In the early days of sanatoria one of the chief ideas was the rest cure, and all types of cases were subjected to it, but as experience increased and knowledge of the disease grew, the treatment varied considerably. It is now recognised that each individual should be dealt with as a separate entity and that mass treatment should be avoided. This mass treatment is the stumbling-block of many institutions, and indeed is the great drawback to sanatorium life generally.

The essential feature of treatment in a sanatorium may be summed up as follows: The patient is placed as far as possible in ideal, hygienic surroundings. His diet is carefully suited to his requirements. Rest, exercise, and work are carefully prescribed. In other words, it is a life lived according to medical prescription.

*Rest Treatment.*—When the patient is first admitted to the sanatorium he should be kept in bed for a few days. This preliminary rest in bed enables him to be more thoroughly under observation, and permits him to recover

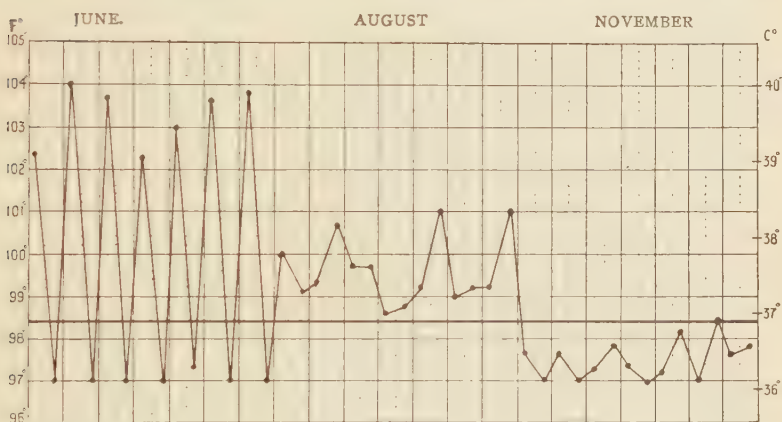


FIG. 45.—Effect of rest in bed on temperature.

from the effects of the journey to the institution, if that should be at some distance from his home. During this time his temperature and pulse will be accurately recorded, and a careful general examination made. If fever be present, and the temperature reach 100° F. in the morning, this period in bed should be extended indefinitely. The patient should be treated much as a typhoid patient would be.

When the morning fever disappears, but fever is present in the evening, the patient may be permitted to rest out-of-doors for the greater part of the day. Attention should be directed to the posture of the "resting-out" patient. The reclining chair should be comfortable, with a sloping back, so that the patient's trunk, shoulders, and head are elevated



at an angle to suit the individual. The back of the chair should be adjustable to any position. The feet of the patient should be elevated from the ground and be practically on a level with the hips. This position assists the circulation of the lower extremities, avoids to some extent that discomfort of the resting-out stage, namely cold feet, and prevents chilblains of the toes. In cold weather special wraps may be used to conserve the heat of the feet and legs, and, if necessary, the patient should have a hot-water bottle. The upper part of the body must be suitably wrapped up, with protection for the ears and thick gloves for the hands.

The situation of the patient when resting out is of importance. He should be so placed that he is not exposed to strong winds or rain, and in winter he should have all the sunshine available. In the height of summer some protection from the sun is advisable.

*Cases suitable for Rest.*—As a routine measure all new patients should pass through a longer or shorter period of rest treatment. Wherever emaciation is marked, or where there has been recent rapid loss of flesh, the resting-out period should be extended until the patient has regained a considerable amount of the lost weight. A patient in whom dyspnoea is a prominent feature should be kept resting. Whenever the disease is apparently active, as evidenced by local and general symptoms, the patient should be kept at rest. The temperature is an excellent guide at this stage. In sanatorium practice it is an almost invariable rule that a patient with either a morning temperature of 99° F. or an evening one of 100° F. (rectal) should rest all the following day.

Rest should be insisted on where tachycardia is present or is brought on by slight exercise. A pulse of over 95 while the patient is at rest is an indication that he should spend the greater part of his day in a reclining chair, and the effect on the pulse of any exercise prescribed should be carefully observed. In some cases of afebrile tachycardia it is good practice to begin, very gradually and cautiously, carefully regulated exercises. These may improve the tone of the heart muscle and thereby lessen the tachycardia.

*Necessity for Supervision.*—When a patient is ordered to rest out, it is necessary to see that this is actually done. If supervision is not carefully exercised, it will be found that the “resting-out” patient rests when it suits him and then spends a considerable part of his day wandering around or standing about idling with other patients. He has an idea that resting in bed or reclining all day in a chair will weaken him. It must be pointed out to him that this is not so. The basal idea of rest is to conserve strength and energy when resting, and it should be explained that wandering around or standing about with other patients is making demands on him which, in his present weak condition, he can ill afford. During the resting period the patient should employ his time by reading, conversation, or by quiet games. Noisy conversation or exacting games should be forbidden. This resting-out stage is extremely irksome to a certain type of patient whose education has been so limited that he knows nothing of the joys of reading.

The resting-out stage should not be unduly long. In the earlier days of sanatorium treatment, this period was prolonged for many months. In fact, the treatment was the “rest cure.” The result, as far as the lungs were concerned, may have been quite good, but the general effect on certain of the patients was sometimes disastrous. They were, by the treatment, converted from active energetic individuals into perpetual loafers. All desire for work seemed to have vanished. They got well by resting, and, to keep well, so they argued, they must still rest if at all possible. Thus they passed on through life making rest their first consideration. At times, if the resting period is unduly prolonged, the patient puts on too much adipose tissue and this makes him breathless on exertion and indisposed for effort. He will complain that the disease has interfered with his breathing, whereas it is really the amount of adipose tissue which is at fault. It is well, then, that the patient at the resting stage should be kept carefully under observation and the rest period not unduly prolonged.

*Exercise.*—After the resting-out stage has been passed through, and the patient is deemed fit for exercise, he should begin with short walks. The distance should be carefully

prescribed. The patient should begin with a short walk of a quarter of a mile in the forenoon and one of a similar distance in the afternoon. The pulse rate and temperature should be carefully noted at this time. If no ill-effects are seen the distances can be steadily increased week by week, so that, as strength increases, the patient may before leaving the sanatorium be walking eight or ten miles per day. The walking exercise should be forenoon and afternoon, and the longer walk should be taken in the forenoon. When the patient begins his walking exercises he should be under strict orders that when his walk is accomplished he must rest. If attention is not paid to this, particularly at this juncture in his treatment, he will be found taking his prescribed quarter of a mile for exercise and then one or two additional quarters visiting different parts of the institution, or he will stand about for long periods, which is equally exhausting. Walking exercise can be carried out in all weathers, but in strong winds the distances should be shortened and the pace slowed. There is no advantage whatever in getting the patient's clothes soaked through in wet weather as was the custom in the early sanatorium days. No harm comes of wearing a waterproof. The walk should be taken at such a rate as to avoid breathlessness.

*Graduated Labour.*—Other forms of exercise besides walking should of course be prescribed. This work should be carefully graded from light to heavy. Work performs a double function. It acts as a stimulus to the patient, who, as he passes from light to heavy tasks, concludes that he must be making progress. This cheerful mental condition is much to be desired, and is a most valuable adjunct to any form of treatment. A second function of work is that it fills in the patient's time and prevents ennui and homesickness.

In all well-governed sanatoria the question of providing work is thoroughly considered and placed on an exact therapeutic basis. Work is done indoors or out-of-doors, and is usually graded from light to moderate or heavy. Indoor occupations consist of polishing brasses, cleaning windows, making beds, dusting the room, sweeping the corridors, and blocking the floors. The amount and the variety of the work depend upon the orders of the doctor. In a sanatorium for

the working classes practically all the indoor work should be done by the patients, with the exception of the kitchen, and even there a great part of the work should be done by them.

The outdoor work begins with light, trivial occupations such as picking up the paper which is so apt to litter the grounds; gathering of dead leaves, succeeded by the raking of walks or plots; Dutch hoeing or edging footpaths. Next come grass-cutting and lawn-rolling, digging with a small light spade, then with a heavier, log-cutting and trenching ground: this last form of work takes the patient well into the navvying stage, and it is one of the surprises to visitors to be shown a gang of men with their coats off and sleeves rolled up, busy doing some trenching work, and to be told that these are patients who a few months previously were pale, anæmic, and wasted. The ordering of all this is strictly in the hands of the doctor, but the nurse will supervise the actual work done indoors. The outdoor work is usually under the personal supervision of the doctor, assisted by the gardener. In order to distinguish the patients who are carefully graded in the various exercises, badges of different colour are worn by them: red may indicate resting out; yellow, slight walking exercise; blue, light work; green, medium; and white, heavy work.

The following is a table of graduated labour for women:—

*Grade I.—Indoor.*

Making beds, cleaning boots, light sewing. Walking exercise, half a mile twice a day.

*Grade II.—Indoor; Outdoor.*

Cleaning light brasses, taps, etc., dusting, light blocking. Picking up leaves, papers, and other light rubbish in the grounds. Assisting to carry away rubbish. Walking exercise, one mile twice a day.

*Grade III.—Indoor; Outdoor.*

Light blocking (1 hour), light sewing, cleaning heavy brasses, sweeping shelters, sweeping leaves, raking and hoeing. Grass-cutting (with assistance); other garden jobs requiring a similar amount of exertion. Walking, two miles twice a day.

*Grade IV.—Indoor ; Outdoor.*

Window-cleaning. Scrubbing half a day. Blocking (heavy). Raking, weeding (half a day), wheeling light barrow. Edging, hoeing. Walking exercise, four miles twice a day.

Walking only to be done if no other work can be found. After work is done patient must be outside.

In sanatoria it is usual to insist on a rest hour before the mid-day and evening meals. This is a good custom, although in some institutions the resting idea is carried to an absurd degree, and the patients are forbidden even to read or talk during this hour. There is no sound argument against reading, as it helps to pass the time pleasantly, and nothing is more irksome for some patients than to have to spend a whole hour without mental occupation. Conversation during the rest hour should be severely restricted, as, with a certain class of individuals, it has a tendency to become argument ; or, if humorous patients are together, conversation may run to boisterousness, and so defeat the purpose of the rest hour.

In some sanatoria, hydrotherapy occupies a prominent position. This usually consists of a cold spray, douche, or wave just after getting out of bed in the morning. It is most efficacious, especially if combined with a smart rubbing of the skin with the towel afterwards. Under the combined influence of the spray and the friction with a towel, the skin becomes much healthier in tone and the cutaneous blood supply better. The patient can withstand the cold more successfully, and the liability to contract chills is greatly lessened. As a rule, after the patients have been accustomed to the spray, they prefer it quite cold, but at first it is better to have the water heated slightly. It is remarkable how readily delicate patients go morning by morning, even in cold wintry weather, to the spray, and in what appreciative terms they speak of its effect. Many patients think so highly of the effect of the cold water on the skin that they indulge in what is called a "half wash" before retiring, *i.e.*, they strip to the waist and scrub the upper part of the body well with soap and cold water, and then get it well towelled. This is a good custom to encourage, as patients on their return home may not have a spray bath, but they can all manage to have



a "half wash" at any rate. An important point for the nurse to observe is that the patient must not be allowed to prolong the time of undressing and dressing, as the body temperature is rapidly lowered and there is risk of a chill. The whole life of the patient in a sanatorium has to be planned and regulated for him. This is one of the secrets and advantages of sanatorium treatment. There ought to be individual supervision, and the idiosyncrasies and susceptibilities of each patient should be met as far as possible.

The following may be taken as a fairly average day for a patient in a sanatorium for the working classes:—

A.M.			
7.30	.	.	Patient rises, washes, and dresses.
8	.	.	Breakfast.
9 to 11	.	.	{ Makes beds, cleans wards and shelters. Graduated work and walking exercise.
11 to 12	.	.	
	.	.	Rest.
P.M.			
12 to 1	.	.	Dinner.
1 to 2	.	.	Rest.
2 to 4	.	.	Graduated work and walking exercise.
4 to 4.15	.	.	Tea (afternoon).
4.15 to 5	.	.	Graduated work—a walking exercise.
5 to 6	.	.	Rest.
6 to 6.45	.	.	Supper.
6.45 to 8.30	.	.	Recreation.
8.30 to 9	.	.	Bed.
9	.	.	Lights out—silence.

*Games.*—Games are a necessary part in the routine of the sanatorium, and they should carefully be suited to the individual patient. Violent games should be forbidden, but the quieter or less strenuous should be encouraged. Golf, without the drive, is excellent. Croquet, bowling, gentle cycling or rowing, horseback exercise, etc., can all be indulged in. When, however, the games cause febrile attacks or tachycardia, they should be forbidden. There is no reason why cards, draughts, chess, etc., should not be indulged in. Care should be taken to see that when playing these games the patients are not permitted to sit in a crouched-up position with the chest compressed, nor should they be crowded

closely together. There is no reason why smoking should be forbidden. Indeed it would be useless to forbid it, for patients would smoke in spite of the most stringent order. The author has seen a non-smoking order put in force in a sanatorium, but the only effect it had was to convert the men into very cute law-breakers. In the end, wisdom prevailed and smoking was permitted. In moderation it has no apparent ill effect on the disease, and its deprivation causes considerable hardship to men accustomed to smoke.

*Education.*—As well as making a direct attempt to cure the patient, a further attempt must be made to educate him so that he may know how to conduct his life on hygienic principles after having left the sanatorium. As a purely curative agency the institution has its limitations. Speaking in general terms, 50 per cent. of the patients sent to a sanatorium are dead within four or five years after leaving the institution. This will show that there is a disappointing feature on the curative side of the work. On the educative side, however, great possibilities exist.

Patients should be carefully trained when in the sanatorium how to use, clean, and disinfect their sputum flasks; how to prevent the spraying about of tubercle bacilli when coughing. They should be taught to appreciate the value of a continual supply of fresh air, and to attend to the quantity and quality of their food. They should also receive a course of simple lectures on such subjects as risks of infection, the methods by which those risks can be minimised, and how tuberculosis can be best combated; in short, how to live a hygienic life, and how to minimise the risk of infection to others.

Such patients when discharged, although not cured, are much less dangerous to the community than those who are untrained. Moreover, the great majority of patients from a well-conducted sanatorium when they return home attempt to carry out the principles they have learned while in the institution. They do this often to the discomfort of the other members of the household who love warm, stuffy rooms, and object to what they call new-fangled ideas about ventilation. The educative aspect of the sanatorium is most important, and if it is neglected the greater part of the value of the institution is lost. Unfortunately in some institutions

the training of the patient is disregarded, and such places are at their worst no better than boarding houses, and at their best, hotels.

**Home Treatment *versus* Sanatorium.**—It is quite unnecessary to discuss, as has been frequently done, the comparative merits of the treatment of the tuberculous patient in (*a*) his own home, and (*b*) in a sanatorium. The two are complementary. The majority of patients have to be treated at home. Even when sent to a sanatorium they can only be there a small portion of their life—a few months at most—and then they return to their homes. In the City of Edinburgh we have, approximately, 2500 notified cases of pulmonary tuberculosis, and, at most, have some 300 beds available for this disease. This gives some idea of the importance of home treatment.

The sanatorium is an undoubted boon for the working-class patient living in a crowded part of the city. Nothing can produce a more marked beneficial result than the transference from the smoke-laden air of the city to the clean atmosphere and the country surroundings, and the ordered life of the sanatorium. Even for the well-to-do patient a residence for a short time in a sanatorium is excellent. The training and discipline are all to the advantage of the invalid, and the mental result of the change is most beneficial. A prolongation of residence extending to many months or even years is quite unnecessary. The same or probably better results would be obtained by treatment at home under the direction of a skilled practitioner.

In the home treatment of a patient there should be set apart one room for his exclusive use. If possible it should have a southern aspect. Extra furniture and ornaments should be removed, but there is no reason why the room should be stripped of everything but a chair, a table, and a bed. This room has to be the patient's headquarters for many months, and it should be made as attractive as possible—in keeping, of course, with hygienic requirements. The windows should open top and bottom, or, better, should be made to open inwards or outwards. The bed should be movable, so that at night it can be pushed up to the window, whereas in the daytime it can be taken

into the interior of the room and the patient's reclining chair placed at the window instead. The indications for rest and exercise are similar to those for a patient in an institution. The temperature and pulse are the main guides. The remarks applicable to guidance as to rest and exercise in the sanatorium are applicable, with equal force, in home treatment. Before undertaking the home treatment of any patient, the physician should make it quite clear that the treatment is, necessarily, a protracted matter, and that great improvement in the lung itself is not to be expected from month to month. This will avoid disappointment to the patient and his friends, who naturally expect glowing accounts of progress at short intervals. The physician should also make it quite clear that he must be strictly obeyed in all things down to trivial details of life. Unless he does so he will find that the patient will speedily fall into the way of doing that which seems right in his own eyes.

One of the great advantages of treatment in the patient's home is that he can have his little peculiarities in food studied in a way that cannot be done in an institution. Another is that he can have comforts at home which are not procurable elsewhere. In the early days of sanatorium treatment the patients were subjected to very rigorous treatment. No fires were permitted, and in cold wintry weather some of the patients suffered severely. There is no reason for this exposure, nor is there any virtue in being made miserable by cold. When the patient is in his own home he can have a fire in his sitting-room, and, without detracting one iota from the efficacy of the open-air regime, can add considerably to his comfort, and in many cases to his chances of recovery.

**Diet.**—Fresh air and good feeding are always linked together when the treatment of a consumptive is discussed. Good feeding, however, is apt to be confused with a surfeit of rich, highly-concentrated food-stuffs, and too much of these soon upsets the digestion of most patients. The tuberculous patient is usually one who has lost some weight, or is actually emaciated, and one of the first objects is so to feed him that he will regain this loss and get back to his normal, and, if possible, half a dozen pounds more than the normal.

There is no virtue in the mere fattening of patients. For a time a patient can gain weight, and yet the disease in the lungs be progressing continuously, and then he slowly loses the weight gained. After much experiment and clinical experience it has been found that a consumptive will do best on the diet suited for a normal healthy individual, but that some additional fat, preferably in the form of milk or butter, can be added with advantage.

The "stuffing" of past days has gone, but in the patient who is under weight a judicious shade of extra feeding is at times desirable. In these patients the appetite is often poor and capricious, and they should be watched carefully, as they may content themselves with a bare subsistence allowance. Then it is that super-alimentation is useful. It should be pointed out to the patient that his appetite is not to be the sole guide, but that he must eat with intelligence. A reasonable portion of food should be given at each meal, and an attempt should be made on the patient's part to consume it.

In former days, when we were accustomed to calculate the exact number of calories that were represented by the diet consumed, the conclusion was reached that a diet representing 3500 calories was quite sufficient for a man and one of about 3000 for a woman. These figures represent a generous diet, as will be seen by the following scale which is taken from Bardswell and Chapman's book on *Diets in Tuberculosis*:—

*Breakfast—*

Milk, 1 pint (coffee added for flavour).

Toast, 2 thin slices.

Butter,  $\frac{1}{2}$  oz.

One egg.

Some meat (1 oz.), or a herring.

If porridge were taken less toast would be necessary.

11 A.M.—

Milk,  $\frac{1}{2}$  pint.

*Lunch—*

Milk,  $\frac{1}{2}$  pint.

Bread, 2 oz.

Butter,  $\frac{1}{2}$  oz.

Fish, 2 oz. (or an ordinary sized helping).



Potatoes,  $2\frac{1}{2}$  oz. (two potatoes the size of an egg).

Green vegetables if liked.

Meat,  $2\frac{1}{2}$  oz.

Milk pudding, 5 oz.

*Tea—*

Tea, thin slice of bread and butter (1 oz.), piece of cake (1 to  $1\frac{1}{2}$  oz.).

*Dinner—*

Milk,  $\frac{1}{2}$  pint.

Bread, 2 oz.

Butter,  $\frac{1}{2}$  oz.

Meat,  $2\frac{1}{2}$  oz.

Potatoes,  $2\frac{1}{2}$  oz.

Boiled pudding, 3 oz.

Soup, green vegetables, or dessert if desired.

*Bed-time—*

Milk,  $\frac{1}{2}$  pint.

This represents a caloric value of 3250. The only difference between this and an ordinary diet for a healthy man is the addition of the extra milk and butter.

When a patient has lost flesh a generous diet should be prescribed. This diet should be rich in fats and proteids. The most easily digested, and, to the majority of people, the most palatable fats are those derived from milk. So when it is desired to increase the fat ration this can be done conveniently by ordering butter or cream. It is an easy matter to get an ordinary patient to consume from two to four ounces of butter per day without any digestive disturbance.

When cream is taken, it should always be diluted with milk or taken with puddings or porridge. Cream is more apt to upset the patient than butter-fat. Probably this is due to the ease with which a considerable amount can be taken. The other sources of fat are fat meat, bacon, certain fish, as salmon, herring, etc., cheese and eggs. Care must be taken that the digestive system is not upset by too much fat, as it is easy to produce flatulent dyspepsia, diarrhœa, etc., by a diet too rich in it.

One may see a patient admitted to the sanatorium, thin and wasted-looking, in spite of the fact that he has been taking many raw eggs per day prior to admission. When

these are cut off and other alterations made in his diet, he may begin to gain weight at once. He has been consuming too much fat and thereby interfering with the normal digestive processes.

There is nothing to be gained by feeding an already well-nourished patient on a fatty diet, as adipose tissue *per se* is no criterion of improvement in the lung condition. Moreover, an excessive amount of fat makes the patient disinclined for work, and breathless on exertion. In regard to eggs, there is a tradition amongst the laity that there is some special virtue in consuming them in a raw state. To many patients this is a repulsive way of taking them, and it is doubtful if there is any extra virtue in them when uncooked. Indeed, some state that they are more difficult to digest when raw than when cooked. They are more palatable and equally nutritious when boiled or poached lightly.

In addition to the fat element in the diet, attention must also be given to the amount of proteids. There is no difficulty in obtaining a considerable choice of food rich in proteid. All forms of animal food—mutton, beef, fish, fowl, etc.—supply it in an easily digestible form. Milk and eggs are also rich in it. At one time in the treatment of tuberculosis raw meat was much used, and with considerable success in some cases. It is not so repulsive to take as might at first sight appear. Usually it is given, minced finely, in the form of a sandwich, but it may be taken with potatoes and tomato sauce. From  $\frac{1}{4}$  to  $\frac{3}{4}$  lb. may be taken per day. It has an excellent effect on anæmic patients. One must remember, however, that there is always a risk of tape-worm from eating raw meat.

The feeding of a consumptive patient is often a matter of difficulty. This is especially so in the later stages. In the earlier stages, when the appetite is good, the patient will consume his food with relish. Even in early stages, and especially with patients in an institution, the diet should be so varied as not to be monotonous. The diet sheet should be so ordered that the patients are not able to tell beforehand what the particular fare of any particular day will be. The diet should be akin to that to which the patient is accustomed at home, but should have the fat and proteid content increased.

## CHAPTER XXIII

### TREATMENT—*continued*

TUBERCULIN has been much vaunted as a specific in the treatment of tuberculosis. It has had a prolonged trial but has altogether failed to fulfil the promises to which its introduction gave rise, and has now no claim to be ranked as a specific in the treatment of this disease. It was first introduced by Koch in the year 1890, and its advent was hailed with the greatest enthusiasm: the whole world was waiting for a cure for tuberculosis, and now the cure was forthcoming. A trial of the new remedy, however, had not proceeded far until it began to be whispered about by clinicians that in many instances the patients were made worse by the remedy, and later several deaths were reported as having been hastened by its use. The pathologists, led by Virchow, also began to note that many of the cases after the administration of tuberculin went rapidly from bad to worse and developed generalised tuberculosis. After a comparatively short trial the remedy was looked upon with considerable disfavour, and was regarded as an extremely potent but dangerous drug. It was discarded by the great mass of practitioners, and only a few persisted in its use. Looking back now, it is not to be wondered at that many cases went from bad to worse. The initial dose in those days was approximately  $\frac{1}{2}$  to 1 c.c. of tuberculin. With later experience we now know that this amount should only be administered at the end of a prolonged course of treatment, with gradually increasing doses, so that it might take approximately nine months to attain to a dosage of  $\frac{1}{2}$  c.c. of the pure tuberculin. After having been out of favour for a few years a second wave of enthusiasm for tuberculin came over the profession. This began again in

Germany, but the method of administering this time was different. A beginning was made with infinitesimal doses, and these were gradually increased, week by week, until maximum doses were reached. As usual, results were produced showing the surpassing efficacy of treatment by this method of administering tuberculin. In our own country tuberculin was also advocated by Wright, but he advised that only infinitesimally small doses should be used, and given in contra-distinction to the German method, in practically the same doses every ten days or so. Wright intended to regulate his dosage by the opsonic index, but this was a tedious and unreliable method of attempting to estimate the dosage and was soon given up.

The varieties of tuberculin which have been introduced would almost appear to be endless. There are already at least eighty different preparations which have been placed before the profession at one time or another. They are all similar in this respect, that if given in a sufficient dose they cause a reaction in the infected individual. In view of the number of tuberculins which have been brought forward as cures, it is reasonable to assume that the greater number of them is of little or no value. Every new tuberculin that is brought forward means that its originator has tried many of the previous varieties and found them wanting, and that he now introduces what he thinks is a better remedy.

Tuberculins can be roughly divided into three classes, viz.: those in which the exo-toxins of the bacillus are employed; those in which the endo-toxins are used; and those where both toxins are combined.

(1) **Exo-toxin Tuberculins.**—The best known and the oldest of these is Koch's tuberculin, which was received with such profound and widespread enthusiasm. This is made in the following manner. A virulent culture of the bacillus is inoculated into a sterile glycerine broth medium, and is incubated at 37° C. for a period of six weeks. After this it is carefully sterilised for one hour, then filtered through a Chamberland filter. The resulting filtrate is then concentrated to one-tenth its original volume by evaporation *in vacuo*. The preparation is brownish in colour, with characterised odour, and keeps indefinitely. It contains about

50 per cent. glycerine, 10 per cent. peptones, and the excretions or secretions of the bacilli which are soluble in glycerine.

In tuberculin literature the symbols "T." or "O.T." (*Tuberculin Original*), or "A.T." (*Alt Tuberculin*), are often used to designate this special variety of tuberculin.

Koch never used the tuberculin in its unconcentrated form, but Denys, working on similar lines, applied the term *Bouillon Filtrate* to the unconcentrated tuberculin, and under this title it has had an extended trial in Belgium. This preparation is simply Koch's original tuberculin before concentration.

Similar preparations have been made by Spengler, but he substituted the bovine type of bacillus for the human type, and he produced what he called *Perlsucht Tuberculin* (P.T.). This is practically identical with Koch's original tuberculin in appearance and effect, and, like it, is concentrated to one-tenth of its original volume. The unconcentrated tuberculin which Spengler used is known as *Perlsucht Tuberculin Originalis* (P.T.O.), and is similar to Koch's original unconcentrated tuberculin.

Attempts have been made to get rid of foreign proteid elements in the tuberculin by growing the bacilli on a peptone-free medium, and the result has been that a tuberculin has been placed on the market as *Tuberculin Albumose Free* (T.A.F.). It is stated to be less toxic than some other tuberculins.

(2) **Endo-toxic Tuberculins.**—In his endeavour to get a specific cure for tuberculosis, when the enthusiasm for his first preparation was waning, Koch set about in another fashion and produced a new tuberculin, viz.: *Tuberculin Ruckstand* (T.R.).

He now grew the tubercle bacilli on a solid medium, scraped off the resulting growth, dried and ground the bacilli to powder in an agate mortar. After the bacilli were thoroughly pulverised, so that no whole organism might be found, a certain weight of the dried powdered bacilli was added to 100 c.c. of distilled water. The mixture was centrifugalised, and the clear supernatant fluid discarded. The deposit (*Tuberculin Ruckstand*) was again dried, powdered, mixed with a small quantity of



water, and centrifugalisated again. The supernatant fluid was poured off, and the whole process was again repeated several times until practically no sediment was left. The fluids resulting from all the centrifugalizations, with the exception of the first, were now mixed, and a slightly opalescent fluid was the result. To it was added 20 per cent. glycerine for preservation.

This is the tuberculin known as T.R., and it should contain in each cubic centimetre 2 mg. of solid substance, which represents 10 mg. of the original dried tubercle bacilli. It keeps for a long period without apparent deterioration.

A still further modification was made later by Koch when he produced his *Bacillary Emulsion* (B.E.). This is made by taking  $\frac{1}{2}$  grain of dried, powdered bacilli, adding this to 100 c.c. of distilled water and glycerine, equal parts, and making an emulsion by prolonged shaking. This preparation is practically identical with T.R., except that the preliminary washing with water and the removal of the more soluble toxins is avoided.

**Tuberculin containing Endo- and Exo-toxins.**—The best known variety of this type of tuberculin is that introduced by Béraneck in 1903 (T.Bk.). This is made by growing the bacilli in a 5 per cent. glycerine-bouillon medium, free from peptones. The culture is filtered but not subjected to heat. The bacilli themselves are next exposed to prolonged action by a 1 per cent. of orthophosphoric acid, and equal parts of the filtrate and the phosphoric acid extract are mixed together, forming Béraneck's tuberculin.

The essential unity of all tuberculins appears to be established from the fact that, if given in sufficient dosage, they all produce a reaction in a tuberculous individual, whereas they have no apparent effect on the non-tuberculous. The specificity of this reaction has now generally been accepted, although it has been proven that many proteins will produce a similar effect in diseased individuals. An injection of nucleo-protein or yeast protein, for instance, will produce reactions similar to those caused by the injection of tuberculin.

In selecting any tuberculin for therapeutic purposes it should be remembered that they are not all equally toxic.

Amongst the milder preparations may be reckoned *Tuberculin Ruckstand*, *Béraneck's Tuberculin*, and *Bouillon Filtrate* (T.R., T.Bk., and B.F.). Amongst the more potent are the original tuberculins, whether made from the bovine or the human type, and the *Bacillary Emulsion* (T., P.T., and B.E.).

**Selection of Patient.**—Much care should always be taken in the selection of any patient for tuberculin therapy. Put shortly, the best type of patient desired is an early, non-febrile case, or a non-febrile case which is making little or no progress with other forms of treatment. The selection of these types of cases, however, vitiates every result given. In an early, non-febrile type of case there is often a doubt as to whether the disease is not already arrested, and that we are now attempting to cure a cicatricial condition in the lung. Assuming, however, that this is an early progressive case without much fever, this is just the type of case that should do well under ordinary hygienic measures; and in the case of a patient with definite disease, but without fever, we are probably dealing with fibroid phthisis, where the formation of fibrous tissue predominates over the breaking down of the lung, and, as a general rule, the prognosis for life, in the absence of fever, is fairly satisfactory. In administering tuberculin to these cases little credit can be attributed to the drug if the patient is found to be alive and working so many years after the treatment.

Tuberculin should not be used in cases where fever is present, though some observers have stated that small doses were usefully employed in reducing the temperature. Generally the drug is quite useless for this purpose. Tuberculin is contra-indicated in cases with a tendency to hæmoptysis; in rapidly advancing cases of the disease, or in patients with much systemic disturbance.

In cases of miliary tuberculosis tuberculin is quite useless; yet in these cases which are free from secondary infection one might expect to get satisfactory results, but for the fact that the system is so saturated with tuberculin that it is unable to respond to any additional dose. In any given case of ordinary tuberculosis the symptoms and signs indicate that the patient is suffering from the tuberculin

toxin, and it is a little difficult to accept as a fact that an additional dose of tuberculin will produce any beneficial effect in an organism which is already struggling against this toxin.

Tuberculin is quite useless in glands which have become caseous. In cases of tachycardia, with a pulse running up to about 120 per minute, it is inadvisable to administer tuberculin, as the tachycardia may be the chief evidence of a profound toxæmia. Epilepsy is usually reckoned a contra-indication, but there seems no reason why it should be so.

**Administration.**—Tuberculin can be administered by inunction, as by Moro's ointment. This is a 50 per cent. mixture of anhydrous lanoline and old tuberculin, and is applied by rubbing a piece of ointment, about the size of a pea, well into the skin of the thorax or abdomen.

In this method there is no record as to the amount of tuberculin absorbed. This depends on several factors, such as the thoroughness of the application, the thickness and the part of the skin employed. It is not a method to be recommended.

Tuberculin is usually given subcutaneously, and the interscapular region is the best site for the injection. The back of the upper arm is more convenient, but occasionally it happens that the arm becomes swollen to a moderate degree after an injection. This is never seen in the interscapular area. The injection should be made with due antiseptic precautions. The syringe should be sterile, and the skin at the site of the injection vigorously rubbed with a swab of cotton-wool and ether.

**Dosage.**—One underlying principle in the administration of tuberculin is to begin with such a minute dose as will not provoke a reaction, and then advance so gradually and cautiously that severe reactions will be avoided, and in the end a tolerance to large doses of tuberculin may be established. The initial dose has been fixed for most tuberculins in an arbitrary fashion; indeed, every practitioner appears to settle his own beginning dose. Probably no other remedy has had such a widely divergent initial dose. It varies from .001 c.c. to .000001 c.c. of the old tuberculin, and from .005 to .000001 c.c. of B.E. The most common initial

doses for the various preparations may be given, approximately, as follows:—

Tuberculin.			Initial Dose.
T.	.	.	·0000001 c.c. to ·000001 c.c.
T.R.	.	.	·000001 c.c. to ·0001 c.c.
B.E.	.	.	·000001 c.c. to ·0001 c.c.
B.F.	.	.	·00000001 c.c. to ·0000001 c.c.

**Dilutions.**—In order to get these minute doses, a regular system of dilutions is necessary. This is easily accomplished. All that is necessary is a pipette marked in tenths of a c.c., some diluting fluid, which is usually sterile water, with ·8 per cent. of sodium chloride, and ·5 per cent. phenol in it, and half a dozen bottles of about 15 to 20 c.c. capacity. The pipette and the bottles must be sterilised, and the following procedure adopted:—

Add 9 c.c. of the diluting fluid to each of the bottles, and to the first add 1 c.c. of the original solution of tuberculin, and shake thoroughly. This will give solution No. I., 1 c.c. of which will contain ·1 c.c. of the original tuberculin. Then 1 c.c. of the first solution is added to the second bottle, and so on to the required dilution.

This method is shown here in tabular form:—

Tuberculin.		Dilution.		Solution I.	Dosage.
1 c.c.	+	9 c.c.	=	10 c.c.	1 c.c. = ·1 tuberculin.
Solution I.		Dilution.		Solution II.	
1 c.c.	+	9 c.c.	=	10 c.c.	1 c.c. = ·01 „
Solution II.				Solution III.	
1 c.c.	+	9 c.c.	=	10 c.c.	1 c.c. = ·001 „
Solution III.				Solution IV.	
1 c.c.	+	9 c.c.	=	10 c.c.	1 c.c. = ·0001 „
Solution IV.				Solution V.	
1 c.c.	+	9 c.c.	=	10 c.c.	1 c.c. = ·00001 „
Solution V.				Solution VI.	
1 c.c.	+	9 c.c.	=	10 c.c.	1 c.c. = ·0000001 c.c.

Suppose that we wish to begin with an initial dose of ·00000001 c.c., this is conveniently done by using a Record syringe marked in tenths of a c.c. One-tenth of a c.c. of solution VI. will give the exact dose. The doses are increased in the following way:—If solution VI. is being

administered, then the first dose would be .1 c.c., and the succeeding doses would be .2 c.c., .3 c.c., .5 c.c., .7 c.c., .9 c.c., and then 1 c.c. of solution VI., or what is frequently given instead is .1 c.c. of solution V. Some workers employ the following serial doses:—.1, .15, .2, .25, .3, .4, .5, .7, .8, .9. The same process is continued throughout each solution if no reactions are encountered. When a reaction with a temperature of about 100° F. is produced, it is customary not to increase the dose, but to go back at least one and give a slightly longer interval before the next injection.

As the initial dose is often chosen empirically, so is the maximum dose. The usually accepted maximum doses are:—

1 c.c.	.	.	.	Old Tuberculin.
2 c.c.	.	.	.	T.R.
2 c.c.	.	.	.	B.E.
1 c.c.	.	.	.	B.F.

There are many instances in which the patient reacts at every injection beyond a certain point, and it is a mistake to press beyond this. In other cases the patient does not appear to progress and yet does not show any definite reaction. Here also it is inadvisable to proceed further. Hence it is that the term maximum optimal dose has been introduced to signify, not any definite amount of tuberculin, but that dose beyond which it appears harmful to proceed.

**Frequency of Dosage.**—As a rule, in the smaller doses, the tuberculin is given every three or four days. This interval is continued until the initial dose of solution III. is reached, when the interval is extended to one week. Larger doses may be given about once in ten days. The duration of treatment is indefinite. No fixed time can be stated, but to carry a patient up to maximum doses requires from six to nine months. How long the maximum doses should be continued is a matter on which there is a difference of opinion.

One method was to continue the treatment indefinitely, giving the doses at about fourteen days' interval. Another was to discontinue at the maximum dose, wait for a few



months, and then begin a second course. Another was to discontinue only when sputum and systemic signs of disease have disappeared. Still another method was to continue the injections until the cutaneous reaction disappeared, or until there was no general reaction with the undiluted tuberculin.

**Results of Tuberculin Therapy.**—The whole of tuberculin therapy is based on Koch's work on animals. He found that when a healthy guinea-pig was inoculated with tubercle bacilli, as a rule the wound closed and healed superficially. In from ten to fourteen days, however, a hard nodule appeared which soon broke down, forming an ulcer that persisted until the death of the animal.

In an animal already infected with tuberculosis, a different series of events occurred. The inoculation wound healed, no nodule formed, but around the area of inoculation on the second or third day an area of skin became discoloured and finally necrosis set in. This necrosed tissue was thrown off and a small superficial ulcer left, which usually healed quickly and permanently. Further experiments convinced Koch that a dilute emulsion of dead bacilli injected into the tuberculous animal caused the primary inoculation wound to become smaller and smaller until it finally healed. Later he stated that the same result would be accomplished by using his tuberculin, which did not cause abscesses as the injection of the emulsion of dead bacilli was apt to do.

His theory as to the effect of the bacillus on the tissues was that it produced, among other substances, one which caused a coagulation necrosis. In this necrosed tissue the bacilli were unable to grow, and might finally die out. The action of his tuberculin was, he thought, to increase this necrosed area which would slough off, and if there was an outlet would be ejected, carrying the enclosed bacilli with it. His own description, which is quoted from Hamman and Woolman, is interesting, and worthy of full quotation. He recommended beginning with .001 c.c., and that the dose should be rapidly increased. He describes his results as follows :—

“The action of the substance was in general manifested by an increase of cough and expectoration after the first few doses, followed by a steady decrease, and, in favourable cases,

complete disappearance. The sputum, too, lost its purulent character and became mucoid. The number of bacilli, as a rule, first began to decrease as the sputum became mucoid in appearance. They were temporarily missed, but later were again found from time to time until the sputum completely disappeared. Coincidentally night sweats ceased, the general appearance improved, and the patients increased in weight. All of the patients in the early stages of tuberculosis were free of symptoms after from four to six weeks, so that they could be looked upon as cured. Patients with not too large cavities were likewise greatly improved and almost cured."

If Koch had followed his "cured" cases a little longer he might probably have altered his opinion, and one is tempted to remark that since his day we have read of many similar cures which have been brought forward as proof of the efficacy of some new remedial agent. The original work on which Koch bases his tuberculin therapy has been gravely questioned. Indeed all laboratory investigations tend to show that tuberculin will not cure tuberculosis in animals. Fishberg declares quite bluntly, "There is no record in medical literature that any investigator has succeeded in curing or benefiting a tuberculous animal with tuberculin." It has to be stated, however, that experimental tuberculosis in animals is a totally different thing from the disease in man, and that there is still a fairly large body of medical opinion which claims to achieve results from tuberculin which are unattainable by other methods.

The results given by various investigators are conflicting. In the past, the balance of opinion has been slightly in favour of tuberculin, but it is well to maintain a healthy degree of scepticism in regard to many of the sanatorium figures, as so many factors enter into their production. My own work several years ago led me to a verdict of "not proven" in connection with the claims made for tuberculin, and the present consensus of opinion appears to be against its use.

Tuberculin treatment is not without its risks, as occasionally cases begin to go downward from the date of a tuberculin reaction. When it is given the patient should be under careful supervision, and a daily record of the temperature kept. With a close watch on this, on the pulse

rate, and on the general condition of the patient, the treatment may be carried on by any practitioner of experience and intelligence.

**Non-specific Therapy.**—In the absence of any certain specific in the treatment of tuberculosis, a great variety of remedies have been tried in the hope of achieving success. Some of these have been used in the attempt to destroy the bacillus in the tissues or to neutralise its toxins. Up to the present, it must be confessed that the quest for such a drug has been futile, but it is in this direction that our keenest research should be made. Other remedies have been used to increase the patient's resistance to the disease by improving the nutrition. Still, others have been used to deal with symptoms alone.

In the attempt to destroy the bacillus a great number of antiseptics have been employed. Mercury has been administered in a variety of preparations, and at times a marked improvement may be observed in some patients.

At one time succinamide of mercury was used to a considerable extent subcutaneously, but ultimately it was discontinued, as the procedure was painful and gave no special beneficial results.

Iodine has been given in various forms. The pure tincture may be used, beginning with a dose of 1 minim three times a day and increasing up to 20. Some patients can take these doses without inconvenience, but others are very intolerant to even small doses. Recently some French workers have been administering enormous doses up to 600 minims daily of tincture of iodine in glycerine (1 part of the tincture to 2 of glycerine), and are claiming some measure of success.

The iodides may be given with advantage in cases complicated with syphilis, bronchitis, emphysema or asthma. Small doses (2 to 5 grains) are best. A few years ago an attempt was made to produce nascent iodine in the body by giving a large amount (35 grains) of potassium iodide in a single dose, and then administering a solution of chlorine three times a day. It was supposed that the chlorine, meeting the potassium iodide in the tissues, would combine and liberate nascent iodine which would have a more

powerful effect in destroying the bacillus. This method had a fair trial but it was in the end abandoned. As an inhalation iodine has been extensively used—at times with what appeared to be good effects. Williams was an early and strenuous advocate of the treatment of early phthisis by continuous antiseptic inhalation. A convenient method of giving iodine as an inhalation is to use a simple inhaler of Yeo's pattern. The sponge in the inhaler is kept moist with the antiseptic, and the inhaler worn for about thirty minutes, three or four times a day. This time is gradually increased until it is used continuously.

This method has been used extensively in sanatorium practice, but the glowing accounts of early observers have not been confirmed.

A common mixture for inhalation is :—

R. Tr. Iodi.  
Ac. Carbolici.  
Creosote.  
Spt. chloroformi āā ʒij.

This inhalation has a beneficial effect in laryngeal tuberculosis, alleviating the irritation and distress, but it is a little difficult to see how it can have any curative effect on the tuberculous process in the lung.

Iodoform has been tried in large doses up to 30 grains per day by the mouth, but it has now been abandoned. The intravenous injection of an ethereal solution of iodoform was strenuously advocated by Dewar, who claimed brilliant results. Others tried it extensively but failed to corroborate Dewar's result. The intravenous injection of iodoform is now rarely, or never, used. It has to be confessed that this form of treatment produced a marked psychic effect, as the patient could detect the smell of the ether in the breath shortly after the injection, and assumed that it must be a very potent remedy.

*Arsenic.*—This remedy has long been employed in the treatment of tuberculosis, but it, in all probability, owes its reputation to its tonic effect on the blood-forming organs, and on the nervous system, rather than to any specific effect on the bacillus. It may be looked upon as one

of those drugs which raise the general resistance of the patient, and, in this respect, it is of considerable value. It may be given as liquor arsenicalis in combination with cod-liver oil. Cacodylate of sodium in 2 to 4 grain doses has also been used. The newer forms of arsenical preparation have been tried, but no striking success has been met.

Gallyl has been used intravenously, but there is no advantage over giving the liquor arsenicalis by the mouth. In giving any arsenical preparation, a watch should always be kept for symptoms of arsenical poisoning. Some of the preparations have been known to produce optic atrophy, so that additional care should be taken when using some of the less well-known forms of arsenic.

*Creosote*.—This is another old remedy for tuberculosis, and it is probably safe to say that before tuberculin was introduced no other remedy, with the exception of cod-liver oil, had such a widespread reputation. It is again coming into favour. There seems to be no doubt but that it has a very definite effect in reducing expectoration and in lessening its purulency, but this it does rather by its effect on the extraneous organisms than by any effect it has on the bacillus. As a certain amount of the drug is excreted by the bronchial mucous membrane, this may explain, in part, its beneficial results. It has been stated that the administration of creosote will give rise to febrile attacks like the reaction in tuberculin and even produce focal symptoms, and on these grounds it has been claimed that it has a definite selective effect on the tuberculous process; but further proof is required for this claim.

At times creosote is not tolerated well. It produces gastric irritation, causes flatulence, and upsets digestion. In these circumstances it should not be used. When it is tolerated well it should be given in large doses. It is advisable to begin with small doses (1 to 3 minims), and increase up to 20 or 30 minims three times a day. Some practitioners have administered as much as 240 minims per day. It may be given in the form of capsules, or in cod-liver oil, or in a mixture with glycerine and tincture aurantii. Various derivatives of creosote have been given, and when creosote is not well tolerated they may be



tried. The chief preparations are Guaiacol and Guaiacol carbonate; Creosote carbonate, Guaiacol cinnamate. These preparations are, generally speaking, less likely to give rise to dyspeptic symptoms.

Intra-laryngeal injections of various antiseptics have been used, but without any marked success. A favourite injection is a 10 per cent. solution of menthol in olive oil. This has often a marked effect in lessening the cough, but it has no effect on the pulmonary disease. About a drachm, or at most two drachms, of the above solution may be injected into the larynx at a time, the nozzle of the syringe being passed through the glottis. A little practice is necessary to perform this operation neatly. Intrapulmonary injections of various antiseptics have been tried, but this method of treatment is now quite abandoned.

Surgical interference has been occasionally attempted. When the disease has been confined to the apex, this has been excised. There has been an occasional success, but this only proves what an amazing amount of interference the body can withstand. A moment's consideration would show the futility of such an operation as the excision of the apex of a lung. We never know the exact limitations of the tuberculous process, even in what we call an early stage, and in early stages tuberculosis is often curable by other and less drastic methods.

*Cod-Liver Oil.*—This is one of our oldest remedies employed in the treatment of consumption, but there is nothing in the nature of a specific in it. Its effect is due to the improvement in the general nutrition of the patient from the administration of an easily assimilable fat. It has been stated that the beneficial effect of the oil is due to the presence of lecithides or to the unsaturated fatty acids, but this is not yet fully established and requires further investigation. It is probable that a considerable part of the resulting benefit is due to the richness of the oil in vitamins. The oil may be given pure or made into an emulsion. Many patients who cannot take the former take the emulsion quite readily. In administering any emulsion a definite declared amount of oil should be in it. Cod-liver oil should be given in small doses at first, and gradually increased as

the patient can assimilate it. Begin with one drachm three times a day and increase to one or two ounces. It is seldom that a patient gets beyond, or even up to, this dose. The usual maximum dose is about half an ounce three times a day.

It should not be given to feverish patients nor to those in whom it gives rise to dyspeptic symptoms. Children take it well, especially if it be given in the form of a well-prepared emulsion, and they make good progress on it. It is often with advantage combined with hypophosphites, the common preparation of *syrupus ferri-hypophosphitis* being as good as any.

Glycerophosphates of lime or magnesium have been also employed, and in many cases the patient has shown much general improvement. A good preparation is the *syrupus glycerophosphates co.* (1 to 2 dr.) thrice daily.

In the absence of a definite curative remedy, though the whole pathway is strewn with discarded "cures," much attention is paid to the treatment of symptoms as they arise, and these will now be considered.

## CHAPTER XXIV

### SYMPTOMATIC TREATMENT

**Cough.**—This is often one of the most distressing features in a case of tuberculosis, especially in the later stages of the disease. It is so annoying that the patient and his friends are particularly insistent on something being given to alleviate it.

The causation of cough is a somewhat complex matter. It may be due to the amount of secretion in the lungs; to an inflamed condition of the mucous membrane of the bronchi; to laryngeal or pharyngeal conditions, such as chronic inflammation, or to definite ulceration of the larynx. It may be due to mouth-breathing, nervousness, or habit. Before dealing with a cough it is therefore well to determine, if possible, its exact cause. In the early stages of the disease the cough is, as a rule, slight, and confined mainly to the morning hours. Here it is a natural effort to get rid of the phlegm which has accumulated in the bronchi during the night. It usually subsides after the patient has expelled the secretion which is present. Little or nothing need be done for such a cough. If the mucus is viscid and tenacious, and a considerable effort is required to get it up, then a hot drink, soon after waking, assists greatly. Half a tumblerful of hot water with 10 grains of bicarbonate of soda or 20 minims of spirit of chloroform often has the desired effect. Some patients state that nothing succeeds better with them than a hot cup of tea.

If the cough persists during the day and little or no sputum is expelled, the patient should be warned against the habit of coughing and should be encouraged to repress it as much as possible. It is wonderful how a little determination succeeds in these cases. The cough can be greatly lessened

in force and frequency, and any sputum present comes up much more easily. If the throat is irritable it may be soothed by an inhalation of spirits of chloroform, or by one of creosote and carbolic acid in equal parts.

At times in early apical disease the cough is alleviated greatly by counter-irritation in the supra-spinous fossa or in the clavicular region. This counter-irritation may be produced by painting with iodine or the application of small fly blisters. In the later stages of the disease, especially when cavity formation has taken place, the cough is often paroxysmal in character and accompanied by profuse expectoration. Sometimes the patient knows that by adopting a certain posture he can bring on the cough, with resulting expectoration, and then there will be a prolonged period of freedom until the secretion accumulates again. When there is much irritation of the bronchial or tracheal mucous membrane some sedatives as a rule are requisite. Relief may be procured by small doses of bromide of potassium (5 to 15 grains), either alone or combined with chloral hydrate. The ordinary expectorant remedies may be tried, but, apart from the mental effect, they are not of much avail.

Opium in some form or other has usually to be administered in far-advanced cases of tuberculosis. There is no reason why these cases should be denied the comfort brought about by the use of this drug. It may be given in the form of a pill containing  $\frac{1}{2}$  a grain of opium with  $\frac{1}{4}$  grain of extract of belladonna. When the opium disagrees with the patient, some of the other preparations should be employed. Codeina in the form of the syrup may be given with advantage in doses of 1 to 2 drachms. Heroin in  $\frac{1}{12}$  grain doses is also useful. It may be given in a syrup or in glycerine. In the latter form it is particularly useful— $\frac{1}{12}$  grain of heroin in 1 drachm of glycerine.

When the cough is due to laryngeal irritation the injection of a 10 per cent. solution of menthol is beneficial; when due to pharyngitis it may be greatly lessened by gargling with a warm alkaline solution to dissolve off the catarrhal secretion, or by an inhalation of creosote, terebene, or pine oil.

**Fever.**—This is merely an indication of the activity of the

disease in the chest, and, speaking in general terms, it runs in direct relationship to the activity of the disease: the more acute the local disease the higher the fever.

Slight degrees of fever do not call for any special treatment except rest. When the fever is marked absolute rest in bed should be insisted on, and it is wonderful how this, in some cases, reduces the temperature. This is well illustrated by the underlying chart.

Rest should be taken in the open air if possible. Patients are sometimes removed to the wards of a hospital from the

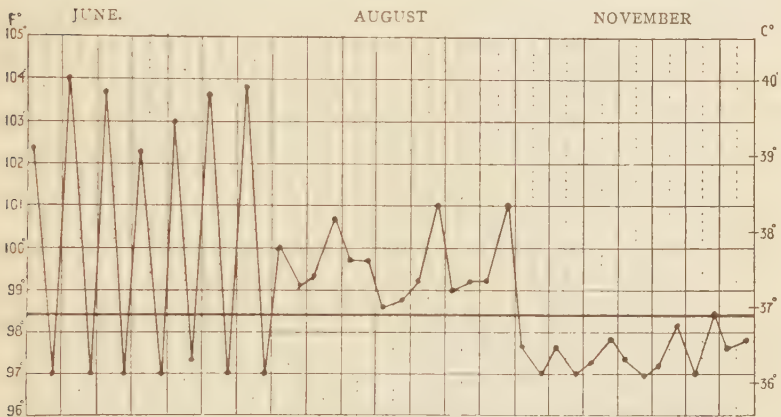


FIG. 46.—Effect of rest in bed on temperature.

open-air shelters because of an acute febrile attack, but this is quite a wrong practice, as it frequently happens that the most beneficial effects on a patient's temperature occur by having his bed removed from the ward right out to the open. Cold air is not harmful, but the reverse.

When absolute rest is insufficient to reduce the temperature, other means must be employed. The indiscriminate administration of antipyretics, as phenacetin and drugs of a similar character, is quite futile. The effect of these is only temporary, and they have no influence on the disease nor on the products of the bacilli. Moreover, they are often followed by marked perspiration and prostration, and produce a depressing effect on the cardio-vascular system.



They should not be employed in routine fashion, but may be given when the patient has headache or muscular pains associated with the fever. When administered they should be given in the usual doses an hour or two before the rise in temperature is expected.

Pyramidon in 5 to 10 grain doses has been highly recommended, but it is no better than antipyrine.

Quinine either alone or combined with digitalis has been extensively used. The quinine should be given in small doses (2 to 3 grains) three times per day. It often acts extremely well. The salicylates or aspirin may also be employed with advantage.

**Night Sweats.**—This is another symptom of a distressing character—especially so when the sweating is profuse. In extreme cases the patient will awake in the early hours of the morning with the nightdress soaked in perspiration. The sweating is often the result of extreme exhaustion in the patient, and not the cause, although patients are apt to attribute their weakness to the profuse perspiration. It is seen in its most marked form in patients who are kept in close rooms with insufficient ventilation and too many bed-clothes. Nothing alleviates night sweats so much as sleeping in the open air or in a thoroughly well-ventilated room, with the bed near the window, and just a sufficiency of light bed-clothes to ensure warmth. Night sweats are rare in sanatorium patients.

Good results are often procured by administering, last thing at night, a glassful of cold milk with the white of an egg switched in. At times the sweating may be controlled by sponging the patient just before bedtime with tepid or cold water, to which some eau-de-Cologne has been added. This method is usually successful where the sweating is the result of high fever. Amongst medicinal remedies which are used with advantage may be mentioned zinc oxide in 5 to 10 grain doses at bedtime, sulphate of atropine subcutaneously in  $\frac{1}{200}$  to  $\frac{1}{60}$  grain doses, agaricine  $\frac{1}{12}$  grain in pill form, and pilocarpine in  $\frac{1}{20}$  grain doses three times a day. When the sweating is the result of nocturnal paroxysms of coughing it can often be controlled by giving opium, in some form

or other, just before bedtime—say, 5 grains of Dover's powder. Whatever remedy is tried, success is more likely to be achieved if attention is paid to the first points laid down, namely, a cool, freely ventilated bedroom and a minimum of light bed-clothes.

**Hæmoptysis.**—In the popular mind blood-spitting is almost always associated with the idea of some exceptional strain. It may be that a violent paroxysm of coughing is blamed, or the sudden exertion due to lifting some extra weight. In some cases such association with stress or strain is close and immediate, but in the majority there is no such connection. In many instances the first intimation of hæmorrhage that the patient has, is the sensation of the saltish taste of blood in the mouth, then he spits and notices the colour. Again, many hæmorrhages take place during the night while the patient is asleep, and he is awakened by the blood coming into his mouth. He spits this out and, in the morning, notices that he has spat up blood. In these patients there is no question of strain, and such cases occur frequently. When the pathology of the disease is considered it will be easily seen that bleeding is quite likely to take place spontaneously, because of the ulcerative process, and without any stress.

The treatment of hæmoptysis is most unsatisfactory because we have no drug which will certainly stop the bleeding. On numerous occasions after administering different drugs, the author has seen the hæmoptysis gradually lessen and cease altogether, and has been inclined to attribute the result to the drug administered; but quite as often the same result occurred when no hæmostatic had been given. Again, drugs apparently successful on one occasion will fail totally on another. It may therefore be concluded that the drugs given had probably little or no effect on the hæmorrhage, and that the cessation was due to natural causes.

In the majority of cases of slight hæmoptysis nothing need be done except to administer some simple placebo for its mental effect. In the sudden profuse hæmorrhages that are sometimes met with, death comes on so rapidly that there is no time to try the effect of any treatment.

There are a few general measures which should be taken in all except the very slightest cases of bleeding. The patient should be put to bed but not laid flat on his back. Here I am in agreement with Fishberg, who advocates a comfortable semi-reclining posture. This is much more agreeable to the patient, as he can be fed more readily, and can expectorate more easily than when lying on his back. It has also a good mental effect, as the patient does not so readily form the opinion that if he but move his head he will cause a recurrence of the bleeding. The bedroom should be quiet, cool, and airy. The diet, in cases where the bleeding is prolonged, should be carefully attended to. The amount of fluid should be curtailed, and the diet restricted to light, easily-digested articles as milk custards, milk pudding, thin porridge, etc. Tea and other stimulants should be avoided. A mouth-wash of weak lemon water is agreeable to the patient. After the hæmorrhage has ceased, the diet should be steadily increased and more solids given, so that, ere a week has passed, the patient can be again on his normal food.

The length of time which patients should be kept in bed is of importance. As a rule, they are kept recumbent too long, but this is better than going to the other extreme. An opinion has been expressed recently that it is unnecessary to keep in bed patients who are suffering from hæmoptysis, and that they do equally well if allowed to walk about as usual—the reason for this line of treatment being that muscular exercise has no effect on the pulmonary blood-pressure. This may or may not be true of the pulmonary artery, but what proof is there that the bronchial artery or one of its branches is not involved? In the meantime, and for some time to come, the traditional treatment of putting the patient to rest in bed will be adhered to.

After the hæmorrhage has ceased for forty-eight hours the patient can sit up safely in bed; at the end of another three days he may be allowed to recline in a chair, and by the end of a week may be allowed a short walk on the level. If the hæmoptysis has been severe, these times should be extended; not so much because of the danger of recurring hæmorrhage, but because of the exhausted condition of

the patient due to loss of blood. Such a patient should not be allowed much exercise for a couple of weeks, and, when not in bed, should be kept at rest in a reclining chair during the greater part of that time. The expectoration of dark clots should not be confused with the actual hæmoptysis. These clots are often expelled for one or two days after the hæmorrhage has ceased.

Looking back over the past score of years, it is interesting to note the drugs which have come and gone in the treatment of hæmoptysis. For slight cases, dilute sulphuric acid in 30-mg. doses every hour or so was routine. The same can be said of gallic acid, tr. hamamelidis, perchloride of iron, and acetate of lead. Weak sulphuric acid is still used, but chiefly on the ground that it is agreeable, does no harm, and has a good mental effect.

For the more severe hæmorrhages, ergot and ergotine were used. The former was given in full doses: one drachm of the liquid extract every hour for eight or ten doses. Ergotine was given subcutaneously. At one time it was almost a neglect of duty not to administer it, and now it is rarely used. Experiences such as these lead one to be somewhat critical of all new remedies which come in with great éclat for the treatment of hæmoptysis or tuberculosis generally. Ipecacuanha in full doses was, at one time, strongly recommended, but this has now been given up and its place taken by emetin. This is given hypodermically in  $\frac{3}{4}$ -grain doses, and can be repeated three or four times a day if necessary. Its use is highly spoken of. The nitrites have long been favourites in cases of severe hæmorrhage. They may be given with advantage when there is a high blood-pressure, thus taking advantage of their vaso-dilator properties. Where rapidity of action is required, nitrite of amyl should be given as an inhalation in 10 to 15 mm. For slower and more prolonged effects  $\frac{1}{2}$  to 2 mm. of a 1 per cent. of nitroglycerine (liquor trinitrini) can be used, or 1 to 2 grains of sodii nitris.

Atropine hypodermically in  $\frac{1}{25}$  to  $\frac{1}{50}$  grain has been recommended, but of this drug the writer has no experience. Supra-renal extract has been used much of late, but the reports of its effects are conflicting. Large doses are stated

to have no effects on the lung circulation, so it is difficult to explain any beneficial effect it may have in hæmoptysis. It may be given in doses of 10 to 30 mg. of adrenalin. Calcium chloride and allied preparations in 10 to 30 grain doses have been much used. At one time they were in routine use in sanatorium practice. Their use depended on their influence on the coagulability of the blood. Recent reports speak well of the intravenous injection of 10 c.c. of a 5 per cent. solution of calcium chloride.

Common salt in doses of  $\frac{1}{2}$  to 1 tablespoonful is an old-time remedy, but it is rarely used now. Van den Velden speaks highly of the intravenous injection of 4 or 5 c.c. of a 10 per cent. solution. The injection of a sterile solution of gelatine has been tried, but this is not without its risks of sepsis, or even tetanus, unless very carefully sterilised. The writer has used it on several occasions, but saw no special beneficial effects. Venesection has been spoken of highly by some observers, but I have no experience of it.

Morphia for a long time now has been a great stand-by in cases of severe hæmoptysis. It calms the patient, allays the cough, lessens the respiratory movement, and in this way undoubtedly lessens the hæmorrhage. It may be given in  $\frac{1}{4}$ -grain doses, but should not be repeated unless at an interval of several hours. It should be remembered that morphia lessens the sensibility of the bronchial mucous membrane, and if too much be given, the blood will not so rapidly be expelled from the bronchioles, and, being retained, adds to the risk of a pneumonia supervening. There are some cases of hæmoptysis so alarming or so continuous that, as a means of arresting it, artificial pneumothorax has to be discussed. Where there is reasonable certainty of the lung from which the hæmorrhage is coming, if pneumothorax be induced on that side the bleeding will quickly cease. In many cases this will arrest what would be, to all appearance, a fatal hæmorrhage. The amount of gas necessary to arrest the hæmorrhage varies. In some cases 250 c.c. have had the desired effect, but in others as much as 1000 c.c. have been required. Usually about 500 c.c. will produce the desired effect. If adhesions are numerous and prevent complete



collapse of the lung, no effect may be produced on the bleeding.

**Gastro-Intestinal Symptoms.**—Loss of appetite, from whatever cause, is one of the great obstacles to the successful treatment of a case of tuberculosis. There is little chance of recovery if the appetite is impaired. Each cause of anorexia must be investigated separately. It may be that it is due to “stuffing” or to too much fat in the diet. For instance, a patient may have been taking too many eggs or too much cream and the extra fat has spoiled his appetite. The correction of any obvious errors in the diet soon alters the state of the appetite. In other instances the loss of appetite is due to lack of proper ventilation in the bedroom, and this is remedied by a thorough open-air regime. In the earlier cases of disease treated in a sanatorium, loss of appetite is not commonly found, and when present is, as a rule, easily remedied.

In the more advanced cases of tuberculosis anorexia is a real difficulty. It may then be due to toxæmia or fever. In dealing with such conditions, attention should be paid to the dietary and the general surroundings of the patient. The food should be such as the patient likes, and it should be daintily served. The more easily digested articles ought to be made use of and the co-operation of the patient enlisted. It should be pointed out to him quite firmly, and yet in kindly fashion, that he must make the attempt to eat, even though he has not his usual appetite, as, with the effort, the desire often returns. Habit in these cases is a great matter. Some of the patients have been under-eating for years, and an effort is necessary on their own part to overcome this. Assistance is often given by prescribing such mixtures as an acid or alkaline vegetable tonic; for instance:—

Rx.	Tr. Nucis Vomicæ.	.	.	℥iss.
	Sod. Bicarbonatis	.	.	℥iss.
	Inf. Gentianæ	.	.	ad ʒvj.

One tablespoonful before meals.

In some patients a marked improvement in the appetite will take place by abolishing supper, and by abstaining from

food for at least three hours before retiring to sleep. Just before retiring, a tumblerful of hot water with 10 grains of bicarbonate of soda dissolved in it should be sipped.

When gastric disturbance with delayed digestion is complained of, the condition is often improved by administering such a mixture as :—

R.	Tr. Nucis Vomicæ	.	.	.	℥ij.
	Vini Pepsinæ	.	.	.	℥iiss.
	Tr. Cardamomi Co.	.	.	.	℥iij.
	Aq.	.	.	.	ad ℥vj.

A tablespoonful after meals.

When the loss of appetite is associated with morning vomiting it may be helped by an alkaline drink when the patient awakes, or by such a mixture as :—

R.	Bismuthi Subnitrat	.	.	.	gr. x.
	Magnesii Carbonatis	.	.	.	gr. v.
	Misturæ Tragacanthæ	.	.	.	℥ij.
	Inf. Gentianæ	.	.	.	ad ℥ss.

Misce. Fiat dose.

Occasional attacks of diarrhœa are common in tuberculosis, but it need not be assumed that they are necessarily all due to tuberculous ulceration of the bowel. These occasional attacks are mostly due to some slight intestinal disturbance caused by unsuitable, or insufficiently digested, food. They require little treatment or, at most, a small dose of castor oil. If a catarrhal condition of the bowel has been set up, this may be improved by a mixture containing bismuth, and by confining the patient to light, easily-digested food for a day or two. When there are disagreeable motions and offensive gases passed, this may be improved by creosote or its derivatives, or by bismuth salicylate in 10 grain doses.

When there is actual ulceration of the bowel the diarrhœa is particularly intractable. Here the diet must be such as will leave no irritating residue in the intestine and should be restricted to milk, custards, raw or underdone minced meat, fish, butter, bread at least one day old, and biscuits. Fried articles should be avoided.

Lime water in ounce doses four or five times per day is sometimes useful. Tannigen 10 grains, bismuth subgallate

10 to 30 grains, or the subnitrate in 10 to 15 grain doses may be used singly, or in combination, and given three or four times per day. For the relief of the occasional pains they may be advantageously combined with some form of opium, say, Dover's powder in 5 to 15 grain doses. In the later stages morphia should be used without hesitation.

Constipation, when present, can be dealt with by attending to the diet and seeing that a fair quantity of vegetables, fruits, brown bread, etc., is taken. A glassful of water last thing at night and first thing in the morning is often helpful. To this water may be added with advantage 20 to 30 drops of the liquid extract of cascara sagrada or a drachm or two of sulphate of magnesia or Glauber's salts.

**Anæmia.**—In some cases of the disease anæmia is profound and calls for immediate attention. In addition to improving the hygienic and dietetic conditions some special treatment is necessary. Iron or arsenic in suitable form is called for. A combination of iron, arsenic, and strychnine in pill form is convenient and easily taken, and extremely good results accrue from such treatment.

Raw meat is very beneficial in this condition and produces a marked and rapid improvement. A residence in a sanatorium without other special treatment also gives excellent results. In a few weeks there is, as a rule, a marked increase in the corpuscle and hæmoglobin content.

**Pain.**—Pain is not a prominent feature in pulmonary tuberculosis, but occasionally it is present to such an extent as to require treatment. It may be due to slight areas of pleurisy, and can be dealt with by mild counter-irritation in the form of Tr. iodine, a mustard plaster, turpentine stupes, etc. When the pain is more severe, the side should be carefully strapped with adhesive plaster. This often gives instant relief. Where the pain is due to muscular strain, the result of coughing, the application of a firm bandage, or strapping the side, gives much relief. This should be combined with some sedative mixture to lessen the cough.

**Insomnia.**—This is not a feature of early tuberculosis

but is sometimes met with in the later stages. It should be treated at first on general lines. The bedroom should be cool, airy, and darkened. There should be an avoidance of company or excitement just before retiring. Tea and coffee should not be partaken of. If these measures are unsuccessful some hypnotics may be employed: sulphonal in 10 to 15 grain doses; veronal, in 5-grain doses, or the bromides may be employed. If the sleeplessness is due to violent paroxysms of coughing, some preparation of opium should be given.

## CHAPTER XXV

### ARTIFICIAL PNEUMOTHORAX

THE treatment of pulmonary tuberculosis, and especially the more advanced cases of the disease, by the production of pneumothorax has come prominently to the fore within recent years. This form of treatment is an attempt to rest the damaged lung, in the hope that reparative processes in the lung will be hastened. It was first advocated by Carson, a Liverpool practitioner, in 1821, but was not then appreciated by the profession, and it is only within the last twenty years, and more especially the last ten, that the treatment has been widely adopted and placed on a scientific basis. In addition to the beneficial effects of the immobilisation of the lung, there are other important changes. The lung, in complete collapse, is reduced to a comparatively airless, fleshy organ. Large cavities, with ulcerating walls, are compressed and the absorptive surface diminished, with the result that there is a very much decreased absorption of the tuberculous and other toxins. The effect of this is seen in the rapid improvement in temperature which sometimes takes place after a complete or even partial collapse.

**Suitable Type of Case.**—Pneumothorax treatment should not be recommended in early cases of the disease. The treatment is not without risk, and it appears inadvisable to expose an early case of tuberculosis to these risks, as such cases are likely to be as much benefited by less severe measures. The ideal case for this treatment is one in which the disease is apparently unilateral, active, and progressive; where there are no pleural adhesions, and the patient is not exhausted by a prolonged illness. Cases of unilateral disease of any extent are, however, rare, as



whenever the disease is present to any great extent in one lung, it is almost certain that the other has become involved, though it may be to a lesser extent. Bilateral disease, however, does not exclude the possibility of pneumothorax treatment, but places a considerable restriction on it, as the lung, which has to do the work of two, must necessarily not be affected to any very great extent. In some cases of acute tuberculosis confined to one lung, the results have been surprisingly good, but, in others, no beneficial results have been noted.

**Contra - Indications.**—Cases of fibroid phthisis are unsuitable for this form of treatment. If the disease is confined to one side, there are, as a rule, firm adhesions in the pleura which prevent collapse of the lung. Indeed in some of these cases the pleural sac is wellnigh completely obliterated. When the disease is bilateral the results are unsatisfactory, as the functioning lung is badly hampered by the increased fibrous tissue. When there is marked emphysema, pneumothorax should not be produced. The presence of disease in the circulatory organs should be looked on as a definite contra-indication, for these patients do not stand the strain at all well. Extensive and severe tuberculosis in other organs, or any disease, such as diabetes or pronounced Bright's disease, should be looked on as a definite contra - indication. Several cases of success in pregnant women have been reported, but before operating in such a case, the risks of the operation inducing premature labour should be discussed, and the patient informed of such risk. It will be thus seen that every case of advanced tuberculosis is not suitable for pneumothorax treatment, and that the operation must not be thought of as a routine procedure. In the past, amongst those who have had a wide experience of this form of treatment only about 2 per cent. to 10 per cent. of cases have been deemed suitable for it. Further experience may increase this proportion.

**Apparatus.**—The articles necessary for this operation are extremely simple, and the simpler they are kept the better.

The essentials are—two glass bottles, some glass and rubber tubing about 4 mm. bore, a manometer, and a perforated needle. In the early days of the operation the

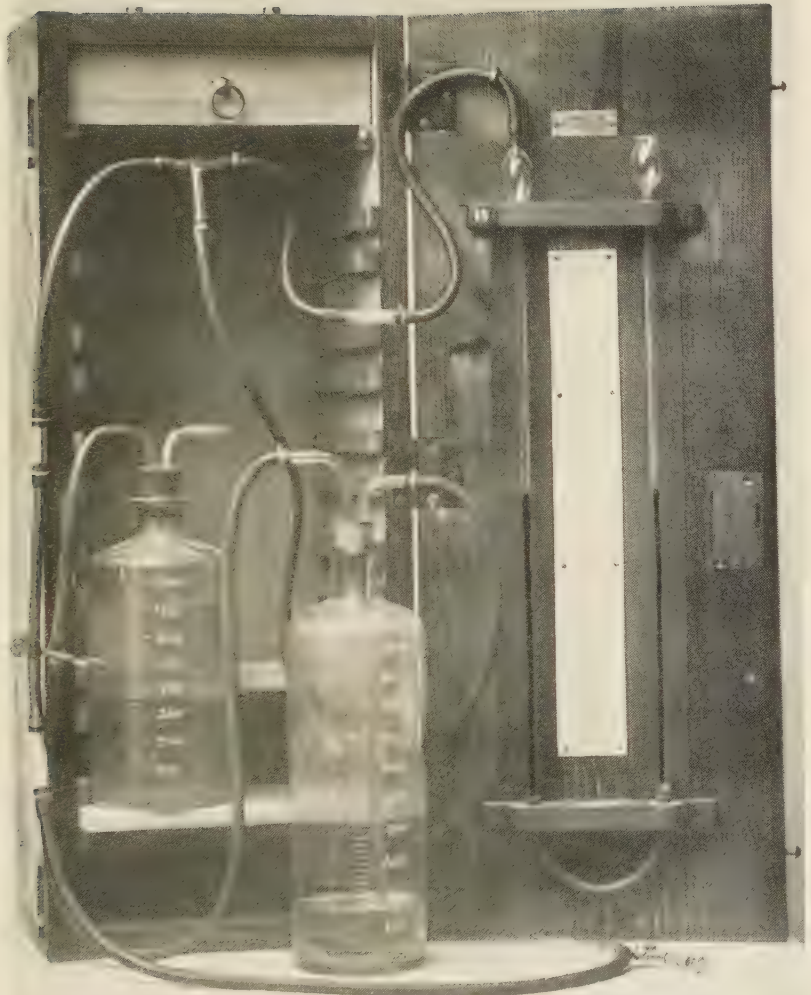


FIG. 47.—Pneumothorax Apparatus.



FIG. 48.—Trochar and Cannula.

[Face page 268.]



gas used was nitrogen, but now filtered air is generally employed as being more convenient. The two glass bottles are so connected with tubing that fluid from one can be syphoned into the other, and the air so displaced from the second bottle is passed through a rubber tube and needle into the pleura. The bottles should hold about 1000 c.c. to 1500 c.c., and one should be so graduated as to record volumes in 100 c.c. They should be movable, so that by lowering or raising one the pressure of gas can be increased or diminished.

Various types of needle have been employed, but that shown in Fig. 48 is probably the most suitable for a first operation. It is really a small trochar and cannula, with an internal bore of about 1.2 mm. For refills (see p. 274) a sharp-pointed needle of about 0.8 mm. bore can be used, as there is then little danger of damaging the lung, which is separated some distance from the pleura.

The manometer is an extremely important part of the apparatus, for the entire safety of the operation depends on it. It consists of two upright glass tubes, about 40 cm. in length, with an internal bore of 4 mm., connected at the bottom by a short piece of rubber tubing. A coloured fluid is poured into the tubes so as to fill them about half full. A scale, marked in half centimetres, is fixed alongside, and so placed that the zero mark of the scale is at the fluid level. The scale is then marked in 1, 2, 3, 4, etc., centimetres above and below the fluid level. The top of one of the glass tubes is widened out somewhat funnel-shaped and left open. The other is connected to the tube leading from the air reservoir to the needle.

*Manometric Readings.*—While the needle is being passed through the parietes there is no alteration in the fluid level; but when it approaches the pleural surface slight oscillations of 1 or 2 cm. may be noted, but the fact that there is no negative pressure should warn the operator that he has not yet reached the pleural sac. When the needle enters the pleural sac there is usually a sharp snap felt by the operator, and at once a distinct negative pressure is registered by the manometer. This pressure may be read on the open limb of the instrument in which the fluid

falls, rising of course to a corresponding height in the other. It should be remembered that only one-half of the pressure is registered on each limb of the manometer. The negative pressure, in a case free from adhesions, may register from 4 to 10 cm., varying with respiration, becoming more marked during inspiration, and lessening during expiration. When these fluctuations are well marked, the gas may be safely allowed to flow into the pleura. At times, after a distinct negative pressure is registered, no oscillations are recorded. This probably means that the lumen of the needle has become blocked, or that it has passed into some soft fibrinous material. When this happens, a blunt stilette should be passed down the needle, and the lumen cleared; if this has no result, the needle should be withdrawn a little. This, in the majority of cases, re-establishes the fluctuation in the manometer and the air may be passed in safely. If no oscillations return, there is some difference of opinion amongst experienced operators as to whether the operation should be continued or not. Some advocate that such a negative pressure means that the needle is in the pleural sac, and the air should be passed in; others state that it is unwise to continue, and that the needle should be withdrawn and a new puncture made, and with this the writer is in agreement.

When there are dense adhesions obliterating the pleural sac, there is no negative pressure and, as a rule, no oscillations, although, occasionally, these may be present ranging from 1 to 2 cm. If the needle enters what is called a pleural pocket, that is, a small area of pleura surrounded by dense adhesions, a negative pressure is recorded as usual; but on passing in a small quantity of gas, less than 200 c.c., the negative pressure changes to positive. In these cases the pressure of the gas which is being passed into the chest may be increased by lowering the bottle containing the air, so that pressures of 5 cm., 10 cm., or even 20 cm., can be used, in the hope that the adhesions may give way and thus produce a more complete collapse of the lung. During an inflation, if adhesions suddenly break down, a positive pressure will immediately give place to a negative one.



If the pleural sac is obliterated, and the needle passes into the lung tissue, the behaviour of the manometer varies according to the tissue into which the end of the needle penetrates. Should it pass into a small bronchiole there is no negative pressure, but slight oscillations, of 1 or 2 cm., take place around the zero mark, the amplitude of the oscillations depending on the character of the respiration. Forced or laboured breathing increases the oscillations, while on a cessation of breathing the pressure returns to zero. Should the needle pass into a small blood-vessel only slight fluctuations occur around the zero mark, a positive pressure being noted during expiration. Again, should the needle pass into a small patch of consolidated lung, there is no change in the manometer, which remains at zero.

It will thus be seen, that for the safe performance of this operation a careful eye must be kept on the various readings of the manometer. A neglect of this may end in disaster.

**Selection of Site.**—The first point to settle before operation, is the site at which the puncture is to be made, and this is determined, in great part, by the location of the disease. In apical lesions, a suitable site is the region of the mid-axillary line, in the sixth or seventh interspaces, or posteriorly, just below the angle of the scapula. These regions are chosen, as there is a minimum of muscular covering on the chest wall and a comparative freedom from adhesions. For basal lesions, a spot may be chosen about the third interspace, external to the nipple line. Evidence of pleural adhesions should always be carefully looked for before the puncture is made, but their presence or absence is a very difficult point to settle. In spite of the dogmatic assertions of some writers, there is always a certain amount of guess-work, and it is only the successful production of pneumothorax which proves the absence of adhesions.

To determine the presence or absence of adhesions, the patient should be carefully examined by X-rays, and the general appearance of the lung and the amplitude of the diaphragmatic excursion noted. A comparatively clear area, unfortunately, does not necessarily mean freedom from adhesions, but it is always a point in its favour. The site should next be carefully percussed, and the ampli-

tude of the excursion of the neighbouring lung edge marked out. A percussion note equal on both sides points towards the absence of adhesions. A good excursion argues in favour of no adhesions, and a limited one for the reverse. Weak or absent breath sounds, as a rule, mean adhesions, but pleural friction is a sign that adhesions have not yet taken place.

While all these points hold good in a majority of cases, there are times when everything points to the almost certainty of adhesions being present, and yet the passage of the needle and the complete success of the operation disproves their presence. On other occasions, when the physical examination would lead one to expect no adhesion, it is found to be impossible to produce pneumothorax at the site selected. No attempt should be made over the site of a cavity. The reason for this will be easily seen.

**Operation.**—The patient should be placed in bed, lying on the healthy side. A small pillow should be placed under the sound side, to arch the body somewhat. The head should be low, and the arm on the side to be operated on, held above the head so as to widen the interspaces.

The area around the site of operation should be covered with sterilised towels, and the skin in the immediate vicinity painted with iodine. If the patient is nervous, a dose of bromide a few hours prior to the operation, or  $\frac{1}{4}$  grain of morphia, subcutaneously, one hour before operating, is helpful. Immediately before the puncture is made, the skin and pleura should be anæsthetised as completely as possible. This may be done by injecting a few drops of a 2 per cent. solution of novocaine into, and under, the skin, at the site where the puncture is to be made. Then the needle, without withdrawing it, is gradually passed through the muscular layer of the interspaces and a small amount of novocaine injected while passing. When the needle reaches the parietal pleura a sharp pain is felt, and the remainder of the novocaine should be injected. In all, about 1 c.c. of the solution is used. The pleura is usually reached at a depth of about 1 to 3 cm., but this depends on the thickness of the parietes. This preliminary anæsthetising should be done slowly, about a minute or so spent over the process. If this is

attended to. little pain should be felt when the trochar is introduced.

After the anæsthetisation is complete, the trochar and cannula are attached to the end of the rubber tubing. Care should be taken to see that there is no moisture in the cannula by placing the instrument in alcohol, and then, before attaching it to the rubber tubing, passing it several times through the flame of a spirit lamp. This drives off the alcohol, and ensures an absence of moisture in the cannula. The bottle containing the air should be empty of fluid, and placed at a slightly higher level than the other. All is now in order to proceed with the operation. The skin, at the chosen site, should be pulled down somewhat, and kept firmly fixed by the fingers of the left hand. The trochar and cannula is then introduced with a firm yet steady, gentle pressure through the skin and subcutaneous tissue to about the depth of 1 cm. At this stage the trochar is withdrawn, and the stopcock turned, so that no air can be admitted. The cannula is now pushed steadily onward, with a slight rotatory movement. When it is passing between the ribs some extra resistance is met at the muscular fascia, but, as a rule, little or no pain is felt at this stage. When the pleura is punctured there is felt a distinct snap, which is sometimes simulated on going through the fascia, but the pleural perforation gives an immediate indication on the manometer by the latter showing a negative pressure and marked oscillations.

The bottle containing the air is now placed on a level with the top of the fluid and a little of the air allowed to flow in at atmospheric pressure. When a small amount has been admitted and the fear of air embolism removed, the gas container can be lowered and the requisite amount of air passed into the pleura. In the early days of the operation as much as 1000 c.c. were admitted at the first operation, but now the consensus of opinion is to limit the amount to about 300 c.c. Where the operation is performed to arrest hæmorrhage, large quantities should be admitted in order to collapse the lung as far as practicable. When the requisite amount of air has been admitted the needle should be slowly

withdrawn. The fold of skin which is pulled up by the cannula is pinched firmly and rolled between the fingers of the left hand so as to obliterate the needle track. The small hole caused by the trochar is sealed with cotton-wool and collodion.

**Refills.**—The first “refill” should take place within three days of the primary operation and in the immediate vicinity of the first puncture. This enables the operator to get more easily into the pleural cavity, as the gas previously injected will not be completely absorbed. If the operation be delayed more than four days, there is the primary difficulty about getting into the pleural sac, and the risk that slight adhesions may have taken place at the seat of the first puncture.

The amount of gas which the pleura will absorb varies a little in different individuals, and varies also whether the patient is in bed or getting about. The amount absorbed at first is from 80 to 100 c.c. per day, but, as the treatment is continued, the pleura absorbs less and less, so that after a few months the amount falls to 50 c.c. or even 30 c.c. per day.

At the first refill it is customary to inject 500 or 600 c.c., but the precise amount is determined by the condition of the patient and the manometric pressure. If there is any dyspnœa, pain in the chest, or disturbance of the pulse, the injection should be limited, and no attempt should be made to raise the pressure above 3 c.c. It is better, if possible, to collapse the lung with the manometric pressure round about zero. If the patient stands 600 c.c. quite well and there is no excessive positive pressure at the end of the refill, not more than about plus 5, then in three or four more days a similar, or, in the absence of contra-indication, an increased quantity up to 800 or 900 c.c. may be injected. If this amount is borne well, in five or six days a similar quantity may be again injected, and so on, until the lung is completely collapsed. After that has taken place, the frequency and amount of the injections depend altogether on the rate of absorption. This is estimated by the physical signs, and more accurately by X-ray examination.

It has been calculated that the adult pleural sac will

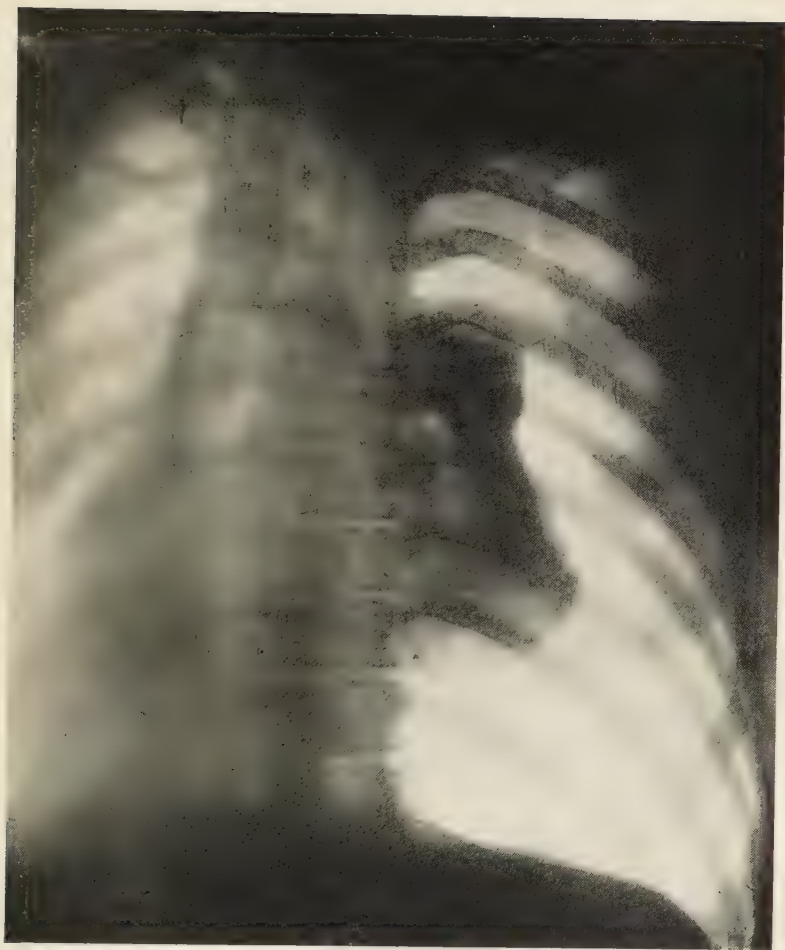


FIG. 49.—Complete Pneumothorax.





contain from 2000 to 3000 c.c. In these early refills the pressure at the end of the operation should not, as a rule, be much above atmospheric pressure, although at times it may reach as much as plus 5. It is better not to introduce the gas at the early refills at a high pressure.

A common experience is as follows:—

	Pressure before Air Introduction.	Pressure after Air Introduction.
1st operation . . .	-8 to -10	-4 to -6
2nd    "   . . .	-6   "   -8	-2   "   0
3rd    "   . . .	-2   "   -4	-2   "   +2

When large quantities of gas are not tolerated well, smaller quantities of about 300 c.c. may be injected every two or three days until total collapse is produced. When this is reached, a positive pressure of 5 to 10 c.c. is sufficient to maintain it.

**Effect on Symptoms and Physical Signs.**—The effect on the symptoms and physical signs of the disease is often very striking. The fever may subside in a remarkable fashion, and with the fever the night sweats disappear. Cough is lessened and sputum definitely diminished. The pulse improved and the appetite increased. (See Fig. 50.) As a rule, the patient will assert that he feels much better. It must not be expected that one, or even two, injections of air will produce all the improvements spoken of. These are seen when further injections collapse the lung. Indeed, after the first few injections, the patient may complain of the increase in sputum, of pain in the chest, and the fever may be unabated. The increase of sputum is due to the collapsing lung pressing the secretion from cavities and bronchi. Perseverance with the treatment often brings its own reward.

In artificial pneumothorax there are none of the extreme symptoms which are often seen in the spontaneous cases occasionally met with in tuberculosis. There is an absence of the acute pain, the cyanosis, the distress, and the collapse.

In the physical signs in the chest there is much alteration in complete collapse, but in partial pneumothorax there is little ; at most, the breath sounds may be slightly weakened.

In total collapse there is some increase in the size of the affected side, with widened interspaces, and some dis-

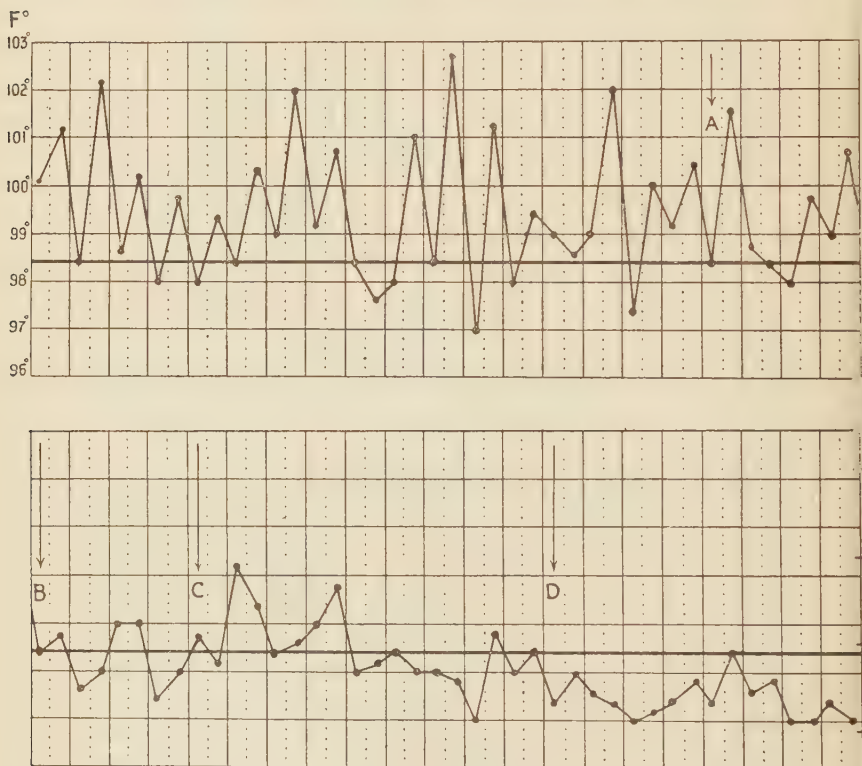


FIG. 50.—Chart showing effect of Artificial Pneumothorax.

A. Initial operation.

B, C, D. Refills.

placement of the apex-beat. The note is occasionally tympanic, but not always so. Metallic tinkling is often heard to perfection. The bell sound produced by striking two coins together, one being placed on the chest wall, is also often heard. Crepitations disappear, and the respiratory murmur is absent; the reappearance of these latter signs being an indication of the re-expansion of the lung.

**Effects on the Lung of Complete Pneumothorax.**—In the compressed lung there is, after some time, a marked proliferation of connective tissue. This tends to the encapsulation and cicatrisation of diseased foci, but in some cases the fibrous tissue development has been so marked that it has prevented the re-expansion of the lung. The development of connective tissue to this extent is, however, rare. The diaphragm, on the affected side, is depressed; it may even be concave upward, and is usually immobilised. Occasionally an inverse action of the diaphragm is seen, that is, the depressed half rises slightly during inspiration, and falls during expiration.

The mediastinum tends to bulge over, to a variable amount, towards the healthy side. This is a point of importance in the treatment; where this tendency is present, the pressure, in the pleura operated on, must not be increased. On the healthy lung there is little effect produced in complete collapse. It has been suggested that a displacement of the mediastinum is apt to bring on œdema of the healthy lung. The warning of the approach of this condition is the appearance of basal crepitations.

**Incomplete Pneumothorax.**—Amongst the cases selected for this treatment there is a considerable number in which it is impossible to induce complete collapse of the lung. Pleural adhesions have been set up by the disease, and these are so firm that they prevent any great degree of collapse. In some cases a small "pocket" of free pleura is found, surrounded by dense adhesions, and in these the injection of a small quantity of gas soon produces a high positive pressure. An attempt to break down adhesions may be made by injecting the gas at a pressure of 15 c.c. to 20 c.c., or even occasionally 30 c.c. At times this succeeds during the operation, and is shown by a high positive pressure suddenly becoming a negative one; at other times, the adhesions give more slowly, and only after frequently repeated injections of quantities of gas sufficient to produce a high positive pressure. The value of partial pneumothorax depends on the extent of the collapse produced. A small, partial pneumothorax is quite useless.

**Duration of Treatment.**—Like all other forms of treat-

ment in connection with tuberculosis, artificial pneumothorax requires to be kept up for a prolonged period. No one limit can be laid down for all cases. It may have to be kept up for one, two, three, or even four years. The length of time depends on the extent and the severity of the original disease. Indeed, there is always a considerable element of guess-work in deciding when the lung may be permitted to expand. The patient should be in fairly normal health, and without constitutional signs of the disease. In order to permit of re-expansion of the lung, all that is necessary is to discontinue the refills. If possible, this should be done in the spring months, so that the patient may have the advantage of the summer when his damaged lung is beginning to resume its function.

The rate of expansion depends on the amount and the extent of fibrous tissue formation in the lung. Assuming that it expands completely, this will be observed, clinically, by the reappearance of the respiratory murmur, and at times fine crepitations and friction. If all goes well, these disappear in a short time, but if the disease undergoes a recrudescence, the crepitations will become moister, and more numerous. The fever, and other constitutional signs, will return. X-ray examination furnishes the best evidence as to the rate of expansion.

Where the lung does not completely re-expand, another problem is presented. Here, the side of the thorax will shrink, producing drooping of the shoulder, and even a slight lateral curvature. The mediastinum and heart will be pulled over, to assist in filling up the empty space, and the diaphragm on the affected side will be drawn upward. There will also be a certain amount of complemental emphysema in the healthy portions of the affected lung. In the cavity, the pressure is much diminished. Cases have been recorded where it reached 40 cm. The patient, as a result of this, may complain of pains and general discomfort in the chest. In these partially expanded cases, in order to avoid the above results, it has been suggested that it is better to keep up, permanently, a partial pneumothorax, the refills being only given at long intervals.

**Results.**—The ultimate results of pneumothorax treat-



ment are difficult to evaluate, as the operation is only performed on patients who, otherwise, have not much chance of recovery.

Riviere, quoting American and European results, gives the following table:—

	American.	European.
	Per cent.	Per cent.
Unimproved, failures, and deaths . . . . .	49·1	42·9
Improved . . . . .	29·2	31·2
Quiescent . . . . .	10·8	9·8
Arrested and cured . . . . .	10·9	16·1

These figures must, of course, be taken with the usual reserve applicable to all sanatorium figures, as the immediate results have often little or no relation to the remote. It has been shown clearly that, in animals, pneumothorax treatment has little influence on the pulmonary lesions.

**Dangers and Complications.**—Artificial pneumothorax is not without its risks. Amongst the chief of these are—air embolism, pleural shock, perforation or rupture of lung, and pleural effusion. Air embolism is one of the accidents of the operation which is becoming rarer, owing to the attention now paid to the manometer. The embolism is the result of puncture of a vein and the entrance of the gas into the circulation, by which it is carried through the left heart to the brain or coronary arteries. It is, however, probable that many cases of so-called embolism are really cases of pleural shock.

The symptoms are sudden collapse and loss of consciousness, irregularity of pulse and respiration, with some patchy cyanosis. Sometimes, before unconsciousness sets in, the patient may complain of giddiness or numbness in hands or feet, or a feeling of pins and needles. The pupils are at times unequal and there may be evidence of hemiplegia. In some cases, death comes on suddenly with immediate cessation of heart action. Air embolism is to be avoided only by careful observation of the manometer, and by being certain, as far as possible, that the needle is in the pleural sac before air is permitted to enter. The treatment of this serious condition consists in lowering the patient's head at once, and injecting some diffusible stimulant.

**Pleural Shock.**—This is an occurrence which takes place,

occasionally, in puncture of the pleura for the withdrawal of pleural effusion, but it is rare. In the performance of a pneumothorax it is undoubtedly more common. This may be due, in some part, to the patient's dread of the operation. The symptoms are similar to those of air embolism, consequently the two conditions may be readily confused. There are varying degrees of shock, ranging from slight faintness to rapid and complete loss of consciousness. The symptoms may come on the moment the pleura is punctured, when the needle is being withdrawn, or even up to half an hour after the completion of the operation. There is pallor, irregularity of pulse, loss of arterial tension, irregularity of respiratory movement, and, in severe cases, loss of consciousness and death.

Pleural shock, while a real danger, does not occur very frequently. Forlanini, quoted by Fishberg, states that he only met it twelve times in 10,000 operations, but Fishberg himself states that he has met it twice in 500 inflations. The prevention of shock is of importance, and this is greatly helped by the administration of a sedative some time before the operation, and by the careful infiltration of the site of operation with a local anæsthetic. It is advisable, also, to administer some diffusible stimulant. During the operation a careful watch should be kept on the patient's pulse, and the operation stopped at once if any irregularity is noted. The treatment consists in lowering the head and shoulders of the patient, and the subcutaneous injection of some diffusible stimulant.

**Perforation of the Lung.**—Perforation or rupture of the lung occasionally takes place. It may be due to actual tearing of the lung tissue by a sharp-pointed needle, or to the accidental rupture of a thin-walled cavity, or caseous patch, in a partially collapsed lung. In some of the cases on record, the rupture took place during exercise in patients who were, apparently, progressing favourably. The symptoms of this accident are sudden acute pain in the side with rapid rise of temperature and the speedy appearance of fluid in the pleural cavity. The fluid is usually purulent, and there is set up a condition of empyema that, in the majority of instances, ends fatally.

**Surgical Emphysema.**—Emphysema of the chest wall is usually the result of using too large a needle, and raising the pressure in the pleural cavity too high. It may be brought about by coughing violently soon after the operation. The condition is easily recognised by a crackling sensation when the fingers are pressed over the swelling. All that is necessary in these cases is to apply firm pressure over the spot where the puncture was made. Occasionally, emphysema takes place into the root of the lung and the posterior mediastinum, and thence spreads up the deep vessels and trachea, when it may appear at the root of the neck. This condition may give rise to some pain in the chest, dysphagia, and even cyanosis, but no permanent harm comes of it. When such emphysema has once arisen, there may be a difficulty in producing complete collapse, owing to the air escaping in this way.

**Pleural Effusion.**—This is one of the most common complications of pneumothorax treatment. Taking all cases together, *i.e.* intermediate and advanced, it occurs in about 50 or 60 per cent.

*Cause.*—Various theories have been advanced to explain the frequency of the complication; the most likely appears to be that prolonged exposure of the pleura to the gas induces a change in the relation of its absorptive and secretory functions, with the result that very little irritation is needed to produce an effusion. This additional irritation may be found in colds, chills, catarrhs, rheumatism. In other cases it is due to the rupture of a small caseous nodule on the periphery of the lung, and the infective material is discharged into the pleural cavity, producing a tuberculous exudate. In a few cases the effusion may be due to an absence of aseptic precautions at the time of performing the pneumothorax.

*Symptoms and Physical Signs.*—There may be no symptoms to draw attention to the effusion. It may be discovered accidentally, by the physician, in his routine examination of the chest, or the patient himself may draw attention to it, by feeling, and hearing, a “splash” in his chest after some jerking exercise. Sometimes the effusion is ushered in with all the signs of a typical pleurisy—pain

in the side, fever, and loss of appetite. If the attack is particularly severe, there may be headache and vomiting.

The physical signs are characteristic. Metallic tinkle is brought out well and easily by getting the patient to shake himself suddenly while the bell of the stethoscope is applied to the chest wall. This will give evidence of fluid so small in quantity as to be detected by no other means. As the quantity increases, there is the characteristic dull note on percussion. It is much easier to detect the upper level in these cases than in an ordinary pleural effusion. The appearance of Grocco's triangle is of little or no importance, and often it is not present.

An X-ray examination is of the greatest assistance. The fluid, which is easily observed, lies at one level, and is not curved as in ordinary pleural effusion. By altering the position of the patient the position of the fluid can be observed to change correspondingly. In some cases there will be noted surface fluctuations, due to the communication of the heart-beat to the fluid. The outlook for the patient is greatly influenced by the character of the effusion—whether it is serous or purulent; whether it is solely due to the tubercle bacillus or to the additional introduction of pyogenic organisms.

The effusion may be serous, slight in amount, and evanescent, or it may slowly increase without any marked constitutional disturbance and still maintain its serous character. These effusions are usually tuberculous, although the bacilli may be difficult of demonstration.

The effusion may be purulent, but this does not necessarily mean that is due to the introduction of septic organisms. Some of the serous effusions become purulent at a later stage, and show no evidence of the presence of organisms other than the tubercle bacillus. Other effusions are quite decidedly purulent from the beginning, and many of these are accompanied by grave constitutional signs from the initial stage. Indeed, there is nothing to distinguish them from the pyopneumothorax due to a ruptured lung. The constitutional signs are marked: high, fluctuating fever, night sweats, rapid emaciation, and a general hectic appearance.

The effect of pleural effusions on the general outlook is varied. Simple serous effusions have, if anything, rather a beneficial effect. They help in the immobilisation of the lung, and may obviate the necessity for frequent refills. Purulent effusions are decidedly harmful. They produce depressing effects on the general health of the patient; they constitute a drain on the system, and they interfere with the re-expansion of the lung. Where operative measures have been attempted for the cure of pyothorax, an open sinus may result, leading to a large suppurating pleural cavity, and the patient may succumb to exhaustion, to tuberculous infiltration of other organs, or to amyloid disease. The risk of purulent effusions constitute a warning against the light performance of a pneumothorax operation.

*Treatment.*—In serous effusions little requires to be done beyond watching the progress carefully. If the effusion fills the whole pleural space, or affects adversely the heart's action, then part of it should be drawn off. If the fluid is withdrawn to relieve the patient of dyspnœa, it is not advisable to refill the space with gas, as the dyspnœa and cardiac distress are apt to return rapidly. Auto-serotherapy has occasionally been practised in these cases. This consists in withdrawing a few cubic centimetres of fluid from the chest and re-injecting it subcutaneously. The reports of the effect of this treatment vary considerably, some writing favourably, others unfavourably, of the procedure. When the effusion has become purulent the pus should be repeatedly aspirated, in the hope that the lung may expand and the cavity be obliterated. Attempts, with varying success, have been made to irrigate the cavity through the cannula with some weak antiseptic, and occasional recoveries have taken place in this way. Surgical treatment, by laying open the cavity, is often a complete failure.



## CHAPTER XXVI

### PROPHYLAXIS

As a fertile source of infection is the sputum of the consumptive, one great point in the prevention of the disease is proper sputum disposal. In an institution this is comparatively easy. The patient is taught to use a sputum flask, and its use insisted on. Much ingenuity has been expended on the production of an ideal sputum flask, but the simpler the flask is the better. It should contain some reliable disinfectant, such as 5 per cent. carbolic solution. In some sanatoria the sputum flask, with its contents, is placed in a special steam steriliser so constructed that the flask can be sterilised and washed out without being handled. After this it is placed to dry, and is again ready for use. In other sanatoria the sputum is mixed with sawdust and burned. This is an unsatisfactory proceeding, and is much inferior to the steam sterilisation. In a private house the contents of the sputum flask, after mixing with an efficient disinfectant, can be emptied with safety into the water-closet.

Male patients, in the interests of cleanliness, should keep their moustaches shaven, or cut extremely short. All indiscriminate spitting should be forbidden, and this rule should be specially observed in workshops and other places where men congregate and spend long hours together.

The consumptive should also be taught the danger there is in coughing, and when in his home or in the company of others should, when coughing, invariably hold his handkerchief in front of his mouth, so as to catch the infected droplets of sputum. This handkerchief can be sterilised by boiling. A special removable and washable lining

should be in the pocket in which the sputum flask or handkerchief is kept. Strict personal cleanliness of mouth, lips, and fingers should be insisted on. The patient should have a bed and, if possible, a bedroom for himself. With attention to these details, and with efficient ventilation in the house, the risks from a consumptive person are reduced to a minimum.

**Prophylaxis in Childhood.**—Probably at no period of our life is it of such vital importance that we should be protected from infection as in infancy. The younger the child is, the more important that it should be protected. Where the father is in an infective condition, this is often a matter of difficulty, but when the mother is infective, the difficulties are wellnigh insuperable. The tuberculous mother should not suckle her infant, should abstain from fondling it overmuch, and should certainly not kiss it. The danger to the child while being breast-fed lies not so much in the risk of tuberculous milk as in the close proximity to the mother while she is speaking or coughing. Tubercle bacilli have been found in breast milk, but not often. The mother should, above all things, refrain from coughing in the vicinity of the baby, and should on no account contaminate its food or feeding utensils by sipping the food before the child has it. Even with the greatest care, it is most difficult to avoid infection unless the mother is prepared to hand over the entire care of the infant to a nurse. In a working-class home this is impossible, so that the chances of a child growing up in such a home are very much diminished. The more serious the state of the mother's health, the poorer is the child's chance of life.

In the case of a consumptive father the chances for the child are better, as the patient is not brought into such close contact with the infant. The author has, however, seen a child, whose father was a consumptive, become the victim of general tuberculosis in spite of every precaution that could be taken by intelligent parents who were alive to the danger the child ran. Thus the risks to a young child, living with an open case of tuberculosis, are great and immediate even although considerable care is taken to avoid them. Apparently the only complete safeguard is immediate and

rigorous segregation, and this is, in the majority of cases, quite impracticable.

For the protection of the child inquiry should be made into the health of other members of the household. The nurse and domestic servants should be healthy. Often the former has undertaken the duties of children's nurse because her own health is delicate. This may be a euphemistic way of stating that she is consumptive. If grandparents live in the house, their health should be inquired into, as very often the so-called chronic bronchitis of the aged is actually tuberculosis.

When the household is free from tuberculosis, there should be little risk of infection to the child if it be kept from contact with tuberculous sufferers. In the case of a bottle-fed baby the milk should, of course, be carefully sterilised, as otherwise it may cause the initial infection, seeing that our milk supplies are infected with bovine tubercle to the extent of from 10 per cent. to 20 per cent.

As a child advances in years the immediate risks to life from infection are not great, although between five and fifteen years of age large numbers of our children have become infected. At this time the death-rate from the disease is at its lowest, but yet no one would willingly expose the child above five to the risks of infection. In school the child should be protected, as far as possible, by the exclusion of any tuberculous scholar, and no tuberculous teacher should be employed.

In the case of the adult the risks from infection are not so great as in childhood, nevertheless no consumptive person should be allowed to work at an indoor occupation which necessitates his being in close and immediate contact with his fellow-workmen. When workmen are sitting around the same bench, or table, day after day with an infectious person, some are almost certain, sooner or later, to fall victims to the disease. It should be noted, however, that in the absence of close, continuous contact the infectivity of the consumptive to other adults is not very marked. Evidence of this is seen in the rarity with which cases of tuberculosis arise amongst nurses and workers in the

disease. A very important point in prevention is the ensuring of efficient ventilation of the factories and workshops where men spend so many days of their lives. In connection with this, it may be remembered that the death-rate amongst men is so much higher than it is in women, and it is probable that the factory life of the man has an important bearing on this fact.

Delicate youths, and especially those of consumptive parents, should be carefully watched during school life and adolescence. As these children are often specially bright mentally and take seriously to studies, no attempt should be made to force them in any way. It is better that they should be given the minimum amount of study and the maximum of physical culture. The choice of a calling or profession is often one of great difficulty. Each case must be dealt with individually. Speaking in general terms, the preference should be for one in which the greatest amount of open-air work can be secured. If a professional life is chosen, then the studies should be proceeded with leisurely and a longer time than usual spent over the curriculum.

## CHAPTER XXVII

### TUBERCULOSIS IN ITS PUBLIC HEALTH ASPECT

FOR the past thirty years or so a very great amount of thought has been given to tuberculosis, and an enormous amount of labour expended on it. This is not to be wondered at when the havoc this disease causes is revealed. It attacks individuals when in the meridian of their physical power, and produces a long-drawn-out invalidism with the consequent heavy drain on the family resources. It contributes largely to the misery of humanity, and to the annual death-rates. Since the discovery of the bacillus there has been an ever-increasing volume of effort to rid ourselves of this disease. In the earlier days the work was begun and carried on by private initiative, and, chiefly as a result of this, public opinion became so educated that at a later date the various bodies concerned with public health became officially interested in the question.

Prior to the year 1906 little or nothing had been done for tuberculosis by the various Local Authorities in Scotland. They were concerned about epidemic diseases, smallpox, scarlet fever, etc., but here was a disease which killed as many persons in a year as all the epidemic diseases did together, and yet no notice was taken of it. In the year 1906 a circular regarding the administrative control of tuberculosis was issued by the Local Government Board for Scotland, in which it was stated that "in no locality have the Local Authority adequately developed the special organisation necessary for the full administrative control of the disease. With a view to assisting in developing and completing their administrative machinery, the Board now direct attention to the following points:—

"I. Pulmonary tuberculosis is an infectious disease within



the meaning of the Public Health (Scotland) Act, 1897." To appreciate the importance and far-reaching character of this pronouncement it is necessary to state here the main provisions of that Act, as modified by the Public Health (Scotland) Amendment Act, 1907, with regard to infectious disease.

- Sect. 45. Gives the Medical Officer of Health power of entry to any house for inspection and examination of suspected house or persons.
- Sect. 46. Gives the Local Authority power to provide disinfecting apparatus for disinfection or destruction of infected goods.
- Sect. 47. Gives power to cleanse or disinfect any house or articles if in the opinion of the Medical Officer of Health this would tend to prevent or check infectious disease. The Local Authority may do it, if the occupier does not do it to the satisfaction of the Medical Officer of Health or a medical practitioner.
- Sect. 48. The Local Authority may, by notice, require the owner of infected bedding, clothing, etc., to deliver it up for disinfection.
- Sect. 51. Any one letting an infected house without disinfecting it, and its contents, is liable to a fine of £20. This section applies to inns and hotels.
- Sect. 52. Any person who lets, or shows a house for letting, and returns a false answer when questioned as to the existence in it of infectious disease within the previous six weeks, is liable to a fine of £20 or imprisonment for one month.
- Sect. 53. Any person ceasing to occupy a house in which there has been an infectious disease within six weeks and who (1) fails to have it and its contents disinfected, or (2) fails to notify the owner or occupier, or (3) denies the fact of the infection, is liable to a fine of £20. Notice to this effect must be served on occupiers of infected houses.
- Sect. 54. Empowers the removal of the infected person if not properly lodged or if in a room with persons other than the necessary attendants.
- Sect. 55. Gives power to retain a person in hospital if he has no place to go where he can take proper precautions against spreading infection.
- Sect. 56. Imposes a penalty (1) on any infected person exposing himself, or anyone else who is infected, in public places, or

(2) on any person selling, pawning, etc. infected goods or washing infected bedding, etc., in public places unless previously efficiently disinfected.

Sect. 57. Imposes a penalty on anyone sending a child to school who has suffered from infectious disease within the previous three months, unless it is medically certified that he may attend without risk of infecting others. Any teacher admitting an uncertified child to school is liable to a penalty of 40 shillings.

Sect. 58. Prohibits, under a penalty, any infected person, or any one living in an infected house, from engaging in any trade connected with handling of food, unless proper precautions are taken against spreading disease.

Sect. 59. Prohibits the carriage of any infected person in any public conveyance under a penalty of £10.

It will be at once seen that the laws for dealing with an infectious consumptive person are far-reaching and powerful, and must be used.

II. The second point dealt with under the 1906 Circular was the importance of the disinfection of the houses inhabited by tuberculous persons, and of the danger of indiscriminate spitting.

III. The third point deals with the provision of hospitals for the treatment of the disease. Hospitals for consumption are classified in the Board's Circular as follows:—

*A.* Curative Hospitals (Sanatoria) for early cases.

*B.* All-day Hospitals. The patients attend the sanatorium during the day and return home at night.

*C.* All-night Hospitals. The patient's work may be suitable for him but his home unsuitable, so he returns to the hospital at night to sleep.

*D.* Convalescent Colonies and Homes. Work Colonies.

*E.* Hospital wards for education, treatment, and control. The patient may be admitted for a short period, and trained in the proper disposal of sputum and methods of preventing infection, and then sent home.

*F.* Hospital wards for isolation of advanced cases.

*G.* Dispensaries for pulmonary tuberculosis.

IV. The fourth section deals with notification of pulmonary phthisis. "For the effective application of the Public Health

Act, notification is essential. The Board will approve of the notification of pulmonary phthisis if they are satisfied that the Local Authority is in a position to deal effectively with the cases notified."

From the foregoing Circular it is clear that the Local Government Board took a wide and comprehensive view of the problem, but many of the smaller Local Authorities were slow to proceed with the schemes. In 1912 pulmonary tuberculosis was made compulsorily notifiable, and two years later this was extended to include all forms of the disease.

In the year 1912 an Inter-departmental Committee was set up to consider the problem of tuberculosis from a national standpoint. This committee, with commendable despatch, issued a preliminary report, in which it recommended what was known as the Edinburgh system. This co-ordinated plan of campaign had been brought into being by the far-sighted policy and indomitable energy of Professor Sir R. W. Philip. The central focus of the scheme was a dispensary, and to this were to be linked a hospital for advanced cases, a sanatorium for early cases, and a farm colony for convalescents who had progressed so far as to be able to resume some out-door occupation.

This machinery, with the exception of the hospital for advanced cases, Sir R. W. Philip had actually brought into operation, and they stand to-day an unsurpassed testimony to his efforts for the combating of this disease. His original diagram deserves copying because of its historic value (Fig. 51).

Partly as a result of the Inter-departmental Report, and partly also because of the offer of a Treasury grant, Local Authorities began to bestir themselves. The grant from the Imperial Treasury was a 50 per cent. one—that is, for every £100 the Local Authorities spend on approved schemes, they could claim £50 as a grant-in-aid from the Treasury. Few Local Authorities, however, even at this date, in spite of this assistance, have anything like a completed scheme. At the most, some have hospital or sanatorium accommodation, and, as a rule, these are insufficient for their needs. Others do not even possess an institution of any sort.

The work in Edinburgh, which may now be detailed shortly, is an integral part of the Public Health administra-

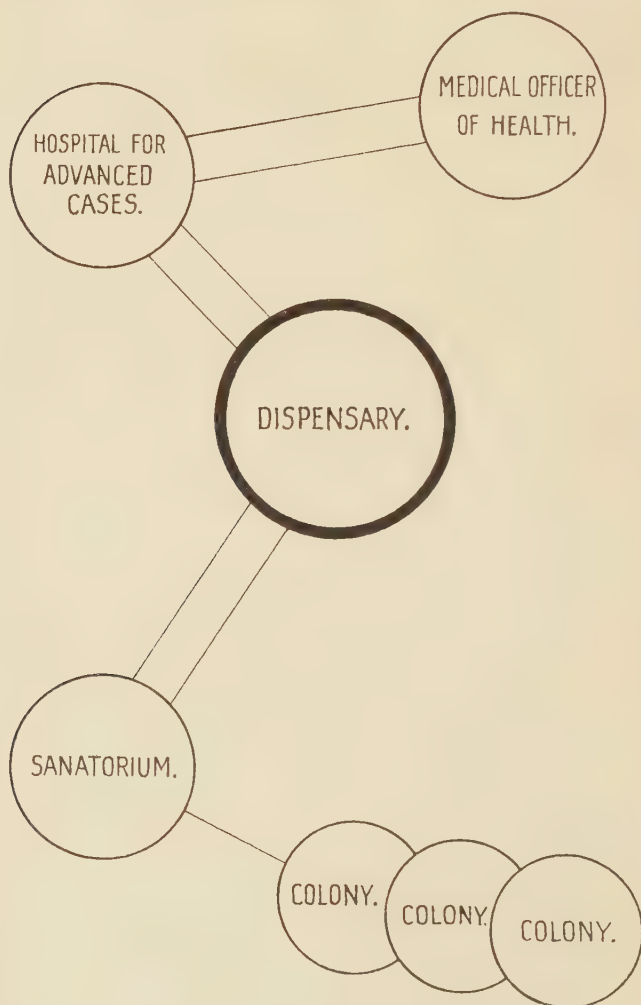


FIG. 51.—Diagrammatic Representation of Tuberculosis Scheme.

tion of the city. The Department for Tuberculosis is under the general control of the Medical Officer of Health, while the actual working of it is under a special Tuberculosis

Officer who ranks on the Public Health Staff as Depute Medical Officer of Health.

As each case of tuberculosis is notified by the medical attendant, it is visited at once by the tuberculosis nurse for the district in which the patient resides.

The city, for tuberculosis purposes, is divided into six districts, with one nurse for each district. The nurses have special training and experience in tuberculosis. The nurse makes a report of the condition and environment of the patient to the Tuberculosis Officer, and visits the patient as often as appears to be required. She takes a general supervision of the hygiene of the house, and instructs the mother, when necessary or advisable, on the economics of housekeeping. She instructs the inmates in the correct method of sputum disposal, and in the steps necessary for the prevention of the spread of the disease. In addition to this, she arranges, as far as possible, for the examination of all the other members of the household, either by their own family doctor or by one of the full-time physicians from the dispensary. The nurse presents a report to the Tuberculosis Officer every quarter on every patient on her visiting list. When a patient is removed to any of the institutions the nurse is at once notified, and on his return home she is again informed. In this way the tuberculous person is shepherded and cared for. Whenever removal to an institution for segregation or curative purposes is indicated either by the medical attendant or by the report of the nurse, the patient is visited by one of the medical members of the Tuberculosis Department and the removal of the patient to the suitable institution arranged for. After removal or after the death of a patient, the room which he occupied and its contents are carefully disinfected.

The dispensary, which acts as a centre to all the other institutions, is conveniently situated and easy of access to all parts of the city. It is open every afternoon for the reception of patients, and is well equipped with dressing and consultation rooms, X-ray installation, laboratory, library, museum, and lecture hall.

Every new patient examined at the dispensary receives literature which indicates in simple language the steps



necessary for the preservation of health and the prevention of tuberculosis, and by this every patient is made a means of spreading the knowledge of the prevention of disease. The extent of the work at the dispensary is indicated in some measure by the number of attendances. These average about 1746 new and 9631 old patients per annum.

The Local Authority has at its disposal in its various institutions over 300 beds. These are apportioned in the following way:—A hospital for advanced cases with 100 beds; a sanatorium for early cases with 130 beds, a sanatorium for non-pulmonary cases of tuberculosis with 64 beds, and a farm colony with 22 beds for the treatment and training of suitable patients with the disease in a quiescent or arrested condition.

The only advances made since Philip drew up his original scheme has been the formation of a village settlement for sufferers from tuberculosis. Such a village is in existence at Papworth, in Cambridgeshire. There the patients with their families form a complete village community. There are village workshops where the patients can follow their own occupations as far as they are able and under suitable conditions. So far no municipality has established a village settlement, but the experiment at present carried on at Papworth will be watched with sympathetic interest. That tuberculosis will ultimately disappear from our midst the writer has little doubt, and if the fall in the death-rate continues as it has done, in another thirty years the part contributed by tuberculosis to the general death-rate will be comparatively insignificant.

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